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CHOROIDAL ANGIO-SCLEROSIS WITH SPECIAL REFERENCE TO ITS HEREDITARY CHARACTER

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(1) Review of Literature

UNDER the name central senile areolar choroidal atrophy, Nettleship described in 1884 a bilateral affection leading to grave impairment of vision. In a relatively sharply circumscribed area extending from the disc to well beyond the macula, varying stages of atrophy of the choroidal vessels could be seen; in the left eye the lesion was almost perfectly circular, in the right rather larger and not quite so regular in outline. The patient was aged 60 years and her sight had been failing for 12 years; her father had been blind six years before he died, but no details were known.

In his report, Nettleship drew attention to a description by Jaeger in 1855. Jaeger's case (described as disappearance of the pigment epithelium of the choroid at the macula lutea) appears to have been unilateral, and as the choroidal vessels are not shown as white lines, Nettleship's case is more definite and more instructive. An almost parallel case was described by Retze in 1902, whose patient, a man aged 66 years, had failing sight for two years and showed the same circumscribed circular area of sclerosis

extending temporally from the disc to well beyond the macula and engulfing it. In both these cases the exposed choroidal vessels revealed almost every range from the normal red colour to conversion into white streaks. A rather similar appearance was reported by Hugh Thompson (1905) in a woman aged 63 years. More recently François (1936) described two such cases.

A case of some interest is described by Knapp under the name of *sclerosis choroidea circinata*.—In a girl aged 11 years an oval area surrounding the macula in each eye assumed the appearance of a *caput medusae* owing to definite sclerosis of the choroidal vessels seen at the periphery of the oval and most marked towards the disc; the macular region itself appeared fairly normal, but the foveal reflex was absent. The family history was negative except for the fact that the grandparents were cousins. The patient herself was healthy. There was night blindness and a scotoma corresponding to the area affected.

No other illustrated descriptions of cases of the kind described by Nettleship and Knapp appear to have been published, but case reports of a number of rather similar conditions are available.

Haab's Atlas contains an illustration of choroidal sclerosis surrounding the disc and extending towards the equatorial region; two somewhat similar cases are illustrated by Di Marzio. Patchy sclerosis near the disc on a background suggestive of mild generalised choroidal sclerosis is pictured by Oeller in healthy patients aged 57 and 71 years. Under the name senile choroiditis Frost's atlas illustrates a case in a man aged 83 years. More extensive lesions are reported by Levinsohn and by Bednarski in whose cases there was widespread, rather patchy, atrophy of retina and choroid in addition to fairly generalised choroidal sclerosis. Levinsohn's patient was a man aged 60 years, of non-consanguineous origin, and healthy apart from his visual defect which had developed seven years before and was associated with night blindness; the family history was clear. Bednarski's case referred to a man aged 23 years, one of a family of 10 of whom 6 died of unknown causes in infancy. The patient developed night blindness at age of 8 years and vision had been failing since. To Bednarski the vascular changes and the rather irregular pigmentary degeneration suggested a mixture of two clinical conditions: choroidal sclerosis and gyrate atrophy of retina and choroid. A case reported by Holloway (1914) appears to have been rather similar to Levinsohn's. More recently Pillat reported two cases of this kind under the name of tapeto-retinal degeneration of the central fundus region. His patients were males aged 42 and 51 years with no history of consanguinity. White dots were present in the periphery of the fundi and were regarded as evidence of retinitis punctata albescens, the central lesion itself

being regarded as central retinitis pigmentosa. Some peripheral loss of field was present in one case.

Sclerosis involving practically the whole of the choroid has been reported by a number of observers. Three plates in Jaeger are highly suggestive, but the first definite case comes from Stanford Morton, whose patient, a man aged 55 years, had full central vision, but a field reduced to 10°. Except for the macular area, the retinal pigment layer and the chorio-capillaris were absent all over the fundus; the sclerosis was most marked towards the disc, the peripheral choroidal vessels appearing normal; pigmentary disturbances were of the slightest. Increasing night blindness and failing vision had been present for about five years. Two plates of rather similar conditions appear in Frost's atlas. A later report came from Bishop Harman, whose patient, a woman aged 57 years, had upward coloboma of the iris in each eye; sight had been failing for 24 years. A case of Thompson (1901) may perhaps also belong to this group. Weiss, reported in abstract, appears to have demonstrated a child with congenital unilateral complete sclerosis of the choroidal vessels.

Aetiological Considerations:

1. *Age.*—Most patients with choroidal sclerosis, no matter of what type, were elderly. But in some cases the history was of long duration, as in Harman's case; Bednarski's patient was aged 23 years and developed night blindness at 8 years; Knapp's patient was aged 11 years.

2. *Syphilis.*—In a case of rather patchy distribution illustrated Oeller (b), and in one of Di Marzio's patients, syphilis appears to have been present. A syphilitic aetiology is suggested in another case of Oeller (a) (1899) and was assumed by Guglianetti for a case of generalised choroidal sclerosis.

3. *Tuberculosis.*—Pulmonary tuberculosis was assumed to be the cause in one of Di Marzio's cases.

4. *Cardio-vascular disease.*—Only in one case (Harman 2nd case) was there a definite heart lesion. Most reports speak of the patient's good health. Morton reported of his patient that seven years later the general condition had deteriorated and that there was albuminuria.

5. *Familial Incidence.*—Most case reports give a negative family history; a familial factor is suggested, but not conclusively, in four case histories.

(i) Holloway and Fewell (1919). In a high myope aged 53 years, whose vision had been poor since childhood, but

who could read till about the age of 27 years, there was generalised choroidal sclerosis, most marked centrally. There was no consanguinity; four brothers living all had poor sight; of six children four survived and had good vision.

(ii) Cuperus (1903). This report is rather more conclusive. In a man aged 70 years, there was choroidal sclerosis radiating from the disc and extending towards the equator. Vision had been failing since the age of 30 years. The parents could see well, but a grandmother died blind. One brother died blind, and another still alive was blind. The patient stated that visual defect in the affected members of the family always developed at about 30 years. According to the report by another ophthalmologist the blind brother alive showed the same fundus appearances as those described by Cuperus for his patient.

(iii) Guglianetti (1908). A man aged 32 years showed widespread choroidal sclerosis with some discrete pigmentary disturbances. The periphery of the fundus was normal. There was no consanguinity and no history of blindness in the family. The patient was subject to Raynaud's disease. According to the report by another oculist, the fundus appearances were the same in a sister, aged 31 years; she was not subject to Raynaud's disease.

(iv) Wilmer (1934). A man aged 35 years with a record of "*retinitis pigmentosa*" for four generations on the maternal side, the mother herself having no eye defect, showed extensive choroidal sclerosis. Pigmentary disturbance was not a marked feature. Judging by the family tree given by Wilmer, the patient had no brothers or sisters and the four ascendants who were supposed to have been blind from retinitis pigmentosa were all males.

In addition to these four groups, two further observations deserve notice:—

(i) Gilbert (1930), in the section on the choroid in the *Kurzes Handbuch der Ophthalmologie* illustrates a case of choroidal sclerosis and states in the text that "familial aspects, hereditary inferiority of the vascular system, are occasionally unmistakable." No further details are given.

(ii) Kapuscinski (1934), reports a family with three affected sibs but normal non-consanguineous parents, showing symptoms suggestive of Friedreich's ataxia, mental deficiency, hypogenitalism together with myopia and choroidal sclerosis

(of the peripapillary type). This group bears some resemblance to the Laurence-Biedl Syndrome and is discussed elsewhere (Sorsby, Avery and Cockayne, 1939).*

6. *Relationship to retinitis pigmentosa.*—In none of the reported cases of choroidal sclerosis was pigmentary disturbance a marked feature, though some clusters of pigment were present in practically all cases. Nor was the appearance of the discs, ranging from normality to atrophy, suggestive of retinitis pigmentosa, though Holloway reports some yellowish pallor of the discs in his case. The retinal vessels were normal in most cases, or showed relatively little narrowing except for marked constriction noted in the case of Holloway and Fewell. Reduced light sense or actual night blindness is noted by most observers. The fields in cases of generalised sclerosis showed reduction to the fixation spot (Morton, Frost) or complete extinction (Harman), but in the cases of localised sclerosis, the field defect corresponded to the affected area, except for some peripheral limitation in the cases of Levinsohn and Pillat.

The possible relationship of choroidal sclerosis to retinitis pigmentosa is further brought out by two families described by Pöllot (1911) and by Di Marzio (1938). Under the name of hereditary atypical retinitis pigmentosa, Pöllot described a boy aged 17 years, showing fairly generalised choroidal sclerosis; his mother had night blindness, and peripheral pigment changes similar to those seen in retinitis pigmentosa. A cousin also showed choroidal sclerosis, and his mother had typical retinitis pigmentosa; a brother was affected with night blindness. Some resemblance to this family with retinitis pigmentosa showing heavy choroidal involvement, is seen in the report by Di Marzio. The patient, a man aged 45 years, had been blind from infancy;

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*(1) Gilbert's observation is now extended by a brief account of choroidal sclerosis in two brothers aged 44 and 45 years. (*Ber. deut. Ophthal. Gesellsch.*, Vol. LII, p. 447, 1938.)

(2) Earlier references to familial choroidal sclerosis appear to be contained in the following observations:—

(*a*) Hutchinson, J. (*Ophthal. Hosp. Repts.*, Vol. VIII, p. 231, 1875.) In his classical account of what is sometimes known as Tay's choroiditis, Hutchinson described amongst other cases a familial group of central lesions in the fundus. These cases have been regarded as the first observations of Doyne's choroiditis (antedating Doyne) and alternatively as cases of macular dystrophy. It is difficult to make a convincing retrospective diagnosis on the description (devoid of illustrations), but it is quite possible that the patients were examples of familial central choroidal sclerosis.

(*b*) Magers in a Thesis, Jena, 1899 (quoted by Leber in *Handb. der ges. Augenheilk.*, Leipzig, 2te Auf., Bd. 7 ii, S. 1208), probably saw a familial group.

(*c*) Haas (*Nederl. Tydschr. v. Geneesk.*, Vol. LXXV, p. 4720, 1931) reports a familial group of macular dystrophy, but his cases undoubtedly represent central choroidal sclerosis. (A man aged 80 years and three of his sons aged 53, 51 and 48 years were affected; two younger daughters aged 46 and 36 years and a son aged 35 years were normal.)

an illustration of the fundus does not show the sclerosis mentioned in the text; a brother and a son are said to be blind. Di Marzio considers this case as one of congenital choroidal sclerosis and an anomalous form of retinitis pigmentosa.

Summary

1. It would appear that three clinical forms of choroidal sclerosis have been described.

(*a*) " Central senile areolar choroiditis " (Nettleship, Retze, Thompson). In this type a more or less regular oval area extends from the disc temporally and engulfs the macula.

(*b*) Peripapillary type (Haab, Harman, Cuperus, Guglianetti, Di Marzio, Pillat). Here the choroidal sclerosis radiates from the disc to a variable but considerable extent peripherally in all directions.

(*c*) Generalised sclerosis (Morton, Frost, Harman). In Morton's and Frost's cases the macula was spared allowing good central vision, with fields of a tubular type. In Harman's case the macula was engulfed.

2. Evidence of syphilis was present in four cases (Oeller, Frost, Guglianetti, Di Marzio); and of tuberculosis in one (Di Marzio). Otherwise the case reports are negative, except that remote consanguinity is noted by Knapp and a possible familial factor is suggested by Holloway and Fewell, by Cuperus, Guglianetti and by Wilmer. In practically all cases, those of Pöllot and of Di Marzio excepted, the diagnosis of " atypical retinitis pigmentosa " could be ruled out.

(In this review the limited peripapillary choroidal sclerosis sometimes seen in myopia and the patchy secondary sclerosis following inflammatory lesions in the choroid have not been considered.)

(2) Case Reports of Familial Choroidal Sclerosis*

(i) " CENTRAL SENILE AREOLAR CHOROIDAL ATROPHY." George R., aged 59 years. Vision: 3/60 each eye. Peripheral fields full. No night blindness. Up to about the age of 20 years was not aware of any eye trouble. Sight has been getting worse progressively till about 10 years ago. A complete medical examination is entirely negative. (W.R.: negative. Electrocardiograph: normal.) Fundi show sharply defined " areolar atrophy " with

* Most of the patients described here have been demonstrated at different times before the Ophthalmological Section of the Royal Society of Medicine. (Meetings of January 1935, December 1936, March 1938, and November 1938).

FIG. 1.

"Central senile areolar choroidal atrophy." Note the extensive choroidal sclerosis and the sharp demarcation of the lesion.

Fig. 2 shows the appearances in the other eye.

Figs. 3 and 4 illustrate the changes seen in the patient's brother.

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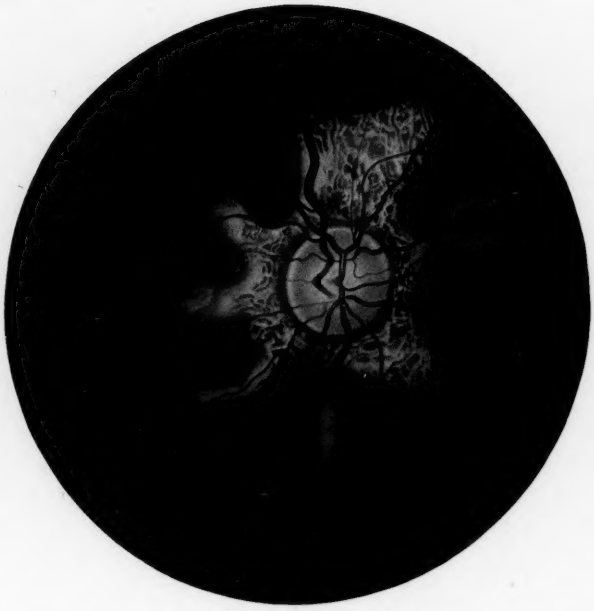


FIG. 5.

Peripapillary choroidal sclerosis. Note the limited distribution of the choroidal sclerosis around the disc. Central vision was unaffected in this eye.

Figs. 6, 7 and 8 illustrate the rather more extensive peripapillary choroidal sclerosis in the other eye, the presence of choroidal haemorrhage, its absorption and the progressive disappearance of sclerosed choroidal vessels.

Figs. 9 and 10 illustrate the more extensive lesion seen in the sister in whom the central areas have become engulfed.

FIG. 2. For legend see Fig. 1.

FIG. 3. For legend see Fig. 1.

FIG. 4. For legend see Fig. 1.

FIG. 6. For legend see Fig. 5.

FIG. 7. For legend see Fig. 5.

FIG. 8. For legend see Fig. 5.

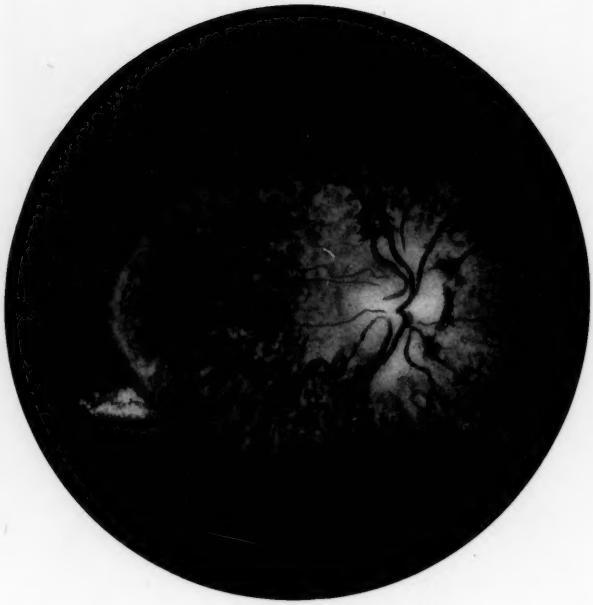


FIG. 9. For legend see Fig. 5.

FIG. 10. For legend see Fig. 5.

FIG. 12. For legend see Fig. 11.

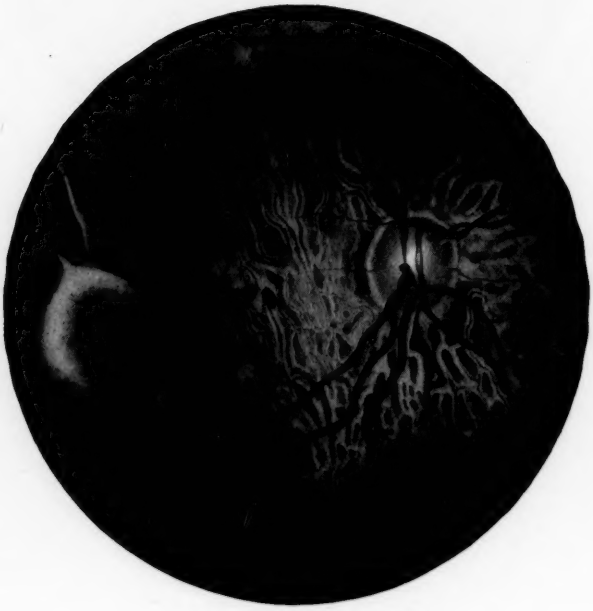


FIG. 13. For legend see Fig. 11.

FIG. 11.

Central and peripapillary choroidal sclerosis. The lesion here bears some similarity to those illustrated in Figs. 9 and 10, at any rate as far as extent is concerned. Yet the lesion in this eye, in the fellow eye shown in Fig. 12 and in the sister's eye (Figs. 13 and 14) does not appear to have originated as a peripapillary choroidal sclerosis extending and engulfing the central area. In these patients the lesion seems to have started in the central area as is shown by Fig. 15 which illustrates the changes seen in 1926 in the same eye as shown in Fig. 11.

FIG. 16.

Generalised choroidal sclerosis. Note the generalised choroidal sclerosis, most marked around the disc. The pigment disturbances bear some similarity to those seen in retinitis pigmentosa, but the disproportion between the vascular and the pigment changes is striking. Fig. 17 shows the appearances in the other eye and Figs. 18 and 19 in those of the patient's sister.

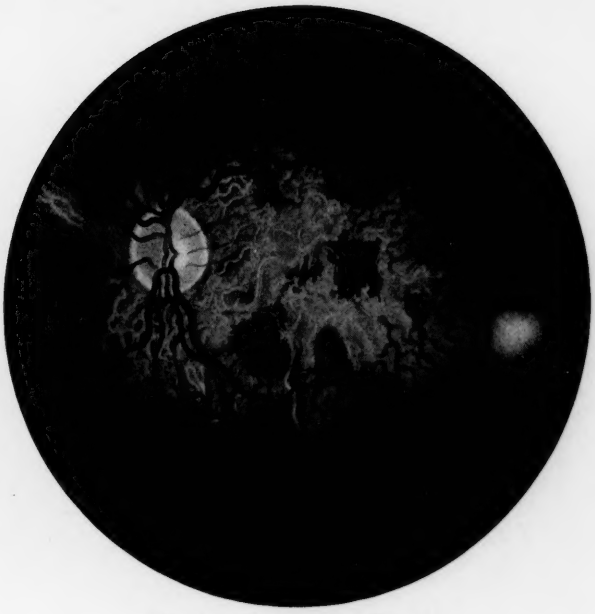


FIG. 14 For legend see Fig. 11.

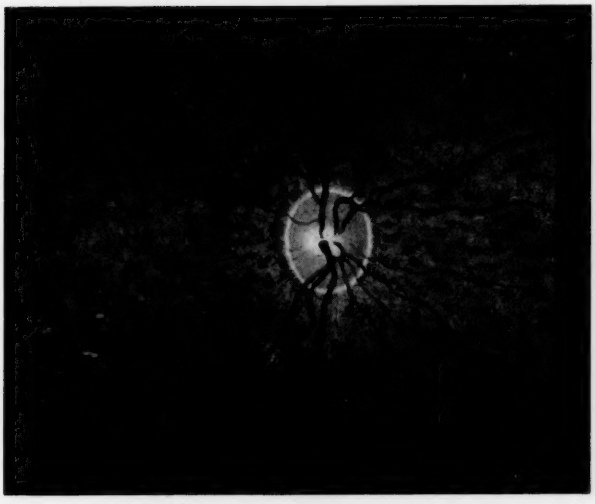


FIG. 15. For legend see Fig. 11.

FIG. 17. For legend see Fig. 16.

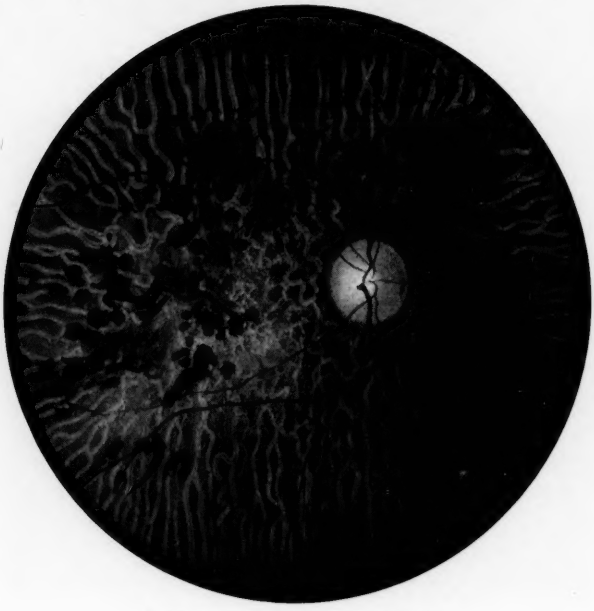


FIG. 18. For legend see Fig. 16.

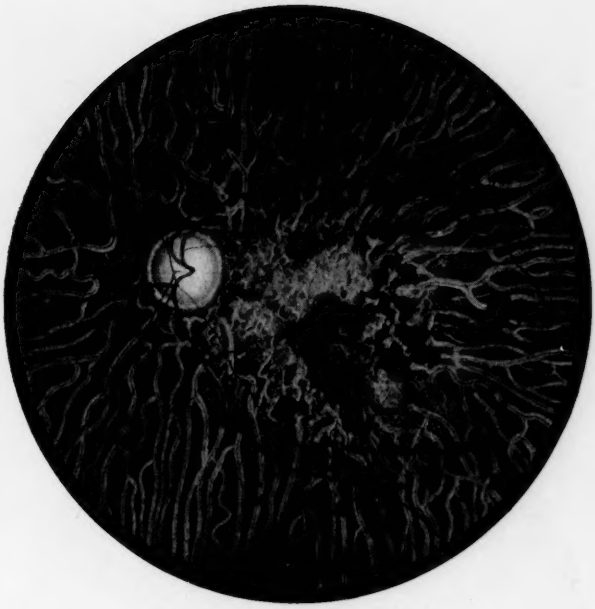


FIG. 19. For legend see Fig. 16.

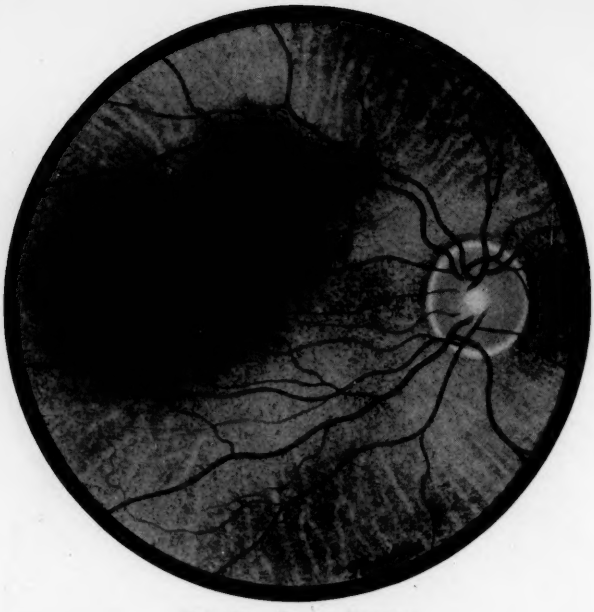


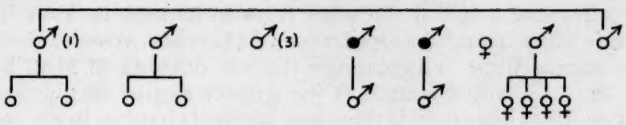
FIG. 20. RIGHT FUNDUS.

FIG. 21. LEFT FUNDUS.

The subjective symptoms in this patient suggested retinitis pigmentosa, but the ophthalmoscopic changes indicate the disappearance rather than sclerosis of the choroidal vessels. An elder brother shows similar fundus appearances and in an elder sister the lesion is confined to the central areas.

some peripheral extension of sclerosis of choroidal vessels (Figs. 1 and 2).

Family history: parents had no eye trouble, and there are no antecedents of any significance. No consanguinity. There were eight children. (1) and (3) are dead. Patient is the fifth member of his family. His immediate elder brother (4) has bad sight, "like himself." All other members of the family are reported



not to have any trouble. The only sons of the patient and his affected brother were examined and found to have normal vision and normal fundi. The affected brother Richard R., whose history is given below, is bedridden, suffering from heart trouble. He was seen at his house and a report on his general condition was kindly supplied by Dr. J. S. Cotman and Dr. Claude Wilson.

Richard R., aged 63 years. Vision: 3/60 each eye. Fields full to hand. No night blindness. Like his brother was unaware of any eye trouble till age of 20 years or so. Sight failed progressively till aged about 50 years. The fundi show essentially the same changes as in his brother, except that the lesion is more sharply circumscribed (Figs. 3 and 4). *General condition:* "When first seen some ten months ago, he had cardiac heart failure (breathlessness, cough, pulse rate 160, liver down to umbilicus). Treatment and rest improved the condition, but the man is a chronic invalid. B.P. 190/110. Heart enlarged; peripheral arteries hardened. Always cyanosed and rather dyspnoeic. No cardiac murmur." *Cardiological report*: "Many premature systoles arising in right ventricle and a few in left ventricle. The normal beats in lead II do not suggest serious myocardial damage, though this is not excluded. Some L.V. preponderance (in axis deviation). On screening, heart not enlarged."

(ii) PERIPAPILLARY CHOROIDAL SCLEROSIS. Mrs. E. N., aged 63 years. Complained of sudden loss of vision of the left eye. Vision Rt: c+1·0=6/5. Left: 1/60, N.I. The peripheral field is full and there is no night blindness. Ophthalmoscopically both eyes showed peripapillary choroidal sclerosis and there was a large central choroidal haemorrhage in the left eye (Figs. 5 and 6). The absorption of the choroidal haemorrhage and the extension of the choroidal sclerosis with the progressive disappearance of the sclerosed choroidal vessels are illustrated in Figs. 7 and 8, drawn respectively four months and eight months later. General examination revealed no abnormalities (B.P. 165/90).

The family history was negative apart from the fact that a sister, Mrs. B., two years older, was said to have been blind for the past three years, and a sister aged about 50 years is said to have no eye trouble. There was no consanguinity. The only daughter of the patient lives abroad and is said to have no eye trouble. The blind sister lives at Hull and through the kind help of Mr. Stenhouse Stewart it was possible to have her fundi drawn. As can be seen from Figs. 9 and 10 they illustrate a more advanced stage of the same features as seen in Mrs. E. N. There is more actual disappearance of choroidal vessels, the condition approaching in appearance the last drawing of Mrs. E. N. (Fig. 8). A marked feature is the greater degree of pigmentary changes. In neither case is there any marked changes in the retinal vessels.

(iii) CENTRAL AND PERIPAPILLARY CHOROIDAL SCLEROSIS. Gertrude C., aged 58 years, and Edith C., aged 59 years. The fundus apppearances, shown in Figs. 11, 12, 13, 14, are essentially similar in the two patients. There is peripapillary choroidal sclerosis, involving the macula and producing heavy pigmentary disturbances. The periphery is clear. Vision : 6/60; the peripheral fields are full and the patients get about quite well. Visual disturbances began in the younger sister fourteen years and in the older ten years ago. The younger sister shows a somewhat raised blood pressure: 175/95; the radial vessels are rather thickened. The elder sister has rheumatoid arthritis and a raised blood pressure: 190/95. The radial arteries are markedly thickened, and exertion brings on shortness of breath. There is no definite heart lesion. The W.R. in both patients is negative.

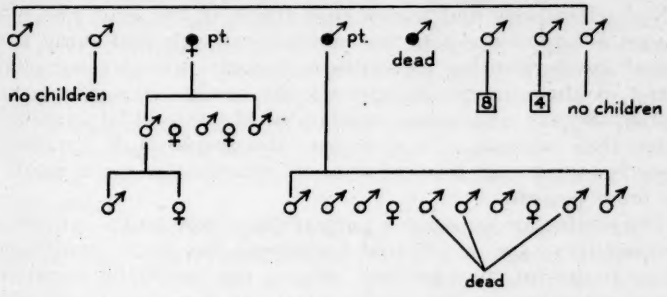
Family history: No consanguinity. The father died at 49 years from " fatty heart," the mother at 64 years from " consumption." There were four children, the patients—the two youngest—are the sole survivors; they are unmarried. An elder sister died at 46 years from " cerebral haemorrhage " and a brother at 41 years from " consumption."

A point of interest is the availability of a fundus drawing at the beginning of the affection in the case of the older sister. She was seen by Mr. F. A. Williamson-Noble in 1926. There was at that time no evidence of choroidal sclerosis and the condition was considered as central choroiditis of possibly tuberculous origin. By courtesy of Mr. Williamson-Noble the fundus drawing is reproduced here (Fig. 15).

(iv) GENERALISED CHOROIDAL SCLEROSIS. (1) Thomas S., aged 60 years, and Mrs. R. L. (a sister), aged 62 years. Vision has been failing since childhood and is now reduced to P.L. There is no definite history of night blindness; in fact, there is a history of sight being better in the evening. Thomas S. is dark haired

and his fundi give a deep colour. Otherwise the appearances are similar to those seen in his sister (Figs. 16, 17, 18, 19), except that the retinal arteries are almost normal in appearance in his case in contrast to the marked narrowing seen in his sister. General investigations, including Wassermann tests: negative.

There are no significant antecedents in the family. The parents were not consanguineous and had good sight. The mother, aged 83 years, is alive. There were 8 children and there is a history of blindness in a younger brother, now dead.



(2) In contrast to this clear cut type of familial choroidal sclerosis, the following case-report illustrates choroidal sclerosis closely simulating retinitis pigmentosa and the end stage is of the type that is erroneously described as choroideremia by some observers.

W. A. C., male, aged 43 years. Parents non-consanguineous. Vision: R. 6/24, L. 6/6. Fields reduced to fixation point. Night blindness. Sight always defective, but became troublesome at about 25 years. The retinal arteries and the discs do not suggest retinitis pigmentosa (Figs. 20 and 21). A brother 11 years older shows the same fundus appearances, except that the arteries are narrow. His vision is 6/9 each eye and the subjective symptoms are so mild that he is barely handicapped. Of particular interest are the ophthalmoscopic appearances in a sister aged 51 years, suggestive of an old central choroiditis. The discs, retinal vessels and periphery are normal. She is not aware of any visual disturbance; her visual acuity is R. 6/6; L. 6/9.

(3) Discussion

Choroidal sclerosis as a clinical entity does not appear to be well established in the literature or in clinical practice. Yet the condition is not particularly uncommon. The older literature does no doubt contain many case reports erroneously interpreted

as the end stage of inflammatory lesions. There are perhaps also records additional to the quoted reports of atypical retinitis pigmentosa which might more properly be regarded as cases of choroidal sclerosis. The review of literature and the personal observations establish choroidal sclerosis as a distinct clinical entity of a familial character. The recognition of the familial character is of value in enabling a study of the different stages of the affection. A family group giving this opportunity has recently been observed by Mr. Frederick Ridley and will be reported in a subsequent paper.

Night blindness and undue contraction of the field was not a feature in any of the patients reported in the present study if the second family showing generalised choroidal sclerosis is excluded —and in these particular patients the fundus appearances suggested atrophy and disappearance of the choroidal circulation rather than sclerosis. In their case the diagnosis of "atypical" retinitis pigmentosa would not be as unwarranted as it would in the other patients.

The distinction between a peripapillary type and a central and peripapillary type of choroidal sclerosis has some justification. Were it not for the previous history, one would be tempted to consider the two patients with central and peripapillary choroidal sclerosis as illustrating a later stage of the affection seen in the two sisters described as showing only peripapillary choroidal sclerosis. Documentary evidence does, however, show that the affection began as a central lesion and that the peripapillary involvement came later.

Just what relationship there exists between these different forms of choroidal sclerosis, is a matter for further study, as are the intermediate stages of these affections and their possible general associations.

The recognition of choroidal sclerosis as a clinical entity raises the problem of the significance of sclerosis of the choroidal vessels in retinitis pigmentosa. The conflicting evidence on the presence of choroidal sclerosis in this affection complicates the issue. The older view that this vascular disturbance was the underlying factor in all cases of retinitis pigmentosa is no longer widely held. A series of observers—Gonin (1903), Stock (1908), Ginsberg (1908), Suganama (1912) and Verhoeff (1930)—have stressed the disproportion between the retinal lesion and the choroidal changes. Ginsberg who emphasised that the cause must be sought in the neuro-epithelium itself, pointed out that the retina may remain intact when there are gross changes in the choroid, as in long-standing glaucoma and nephritis.

The weight of histological evidence is in favour of regarding the neuro-epithelium as the primary seat of the affection, but the

choroidal factor cannot altogether be ignored. Clinically some cases of retinitis pigmentosa show considerable choroidal sclerosis with relatively little retinal involvement, as judged by the pigmentary disturbances and the appearance of the disc. There is, therefore, some valdity in the view advocated by Ascher that retinitis pigmentosa shows two forms, one mesodermal or choroidal, and the other ectodermal or retinal. Some genetic support for this distinction has been advanced by Wibaut. How far these types have a distinct genetic difference and different general associations is still questionable. What is of importance is to recognise that if the choroidal type of retinitis pigmentosa is indeed a clinical entity, it differs considerably from the familial cases of generalised choroidal sclerosis to which attention is drawn in this paper. It is possible that the two familial groups of generalised choroidal sclerosis described here may represent these two types—the S. family being an illustration of choroidal sclerosis and the C. family of the choroidal type of retinitis pigmentosa.

The nature of choroidal sclerosis requires elucidation. The present case reports establish its familial character and the fact that the lesion develops in adult life. Presumably the choroidal circulation earlier in life was normal. In so far as the lesion appears to develop in post-natal life in fully differentiated tissue, and is of a hereditary character, it may be regarded as belonging to the group of heredo-degenerations or abiotrophies.

My thanks are due to Mr. F. A. Williamson-Noble for permission to use Fig. 15, to Dr. J. S. Cotman, the late Dr. Claude Wilson and to Mr. Stenhouse Stewart for the trouble they have taken in supplying information on patients under their care.

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A CASE OF ALEUKAEMIC LYMPHOSIS INVOLVING THE UPPER LIDS. WITH PATHOLOGICAL FINDINGS*

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THE globe and its appendages are occasionally the site of abnormal proliferation of the lymphoid tissues. In very rare cases these occur as local tumour growths, either of benign lymphomatous, or of malignant lymphosarcomatous character. In the vast majority of cases, however, the lesion of this region is merely part of a systemic disease which sooner or later comes to involve more or less of the lymphoid tissue of the body. Consequently, any of the various forms of systemic proliferation of lymphoid tissues may express itself in this region, and hence a great variety

* Read before the American Ophthalmological Society, San Francisco, June 8, 1938.

of these diseases may be the fundamental cause of the tumour-like growth of the eye. In the past, a great deal of stress has been put upon the differential diagnosis of many of these processes from the standpoint that they were to be looked upon as distinct entities. Now we realise that many of these conditions form a related series which differ only in the character of the proliferative activity. Once this fact is appreciated, it becomes quite evident that we are not so concerned with the sharp separation of the various members of this group of diseases, but rather we are interested in their inter-relationship, and the position which the individual case takes in the whole.

A most interesting case of this character has recently come to our attention. It concerns a male patient, aged 65 years, and weighing 236 pounds, who was admitted to the wards of the Royal Victoria Hospital, Montreal, in January, 1938, owing to progressive swelling of both upper eyelids. He first noticed that the lids were swollen the previous July; but this swelling seemed to subside or to be assisted after the use of astringent drops. He was seen at more frequent intervals during the autumn and his admission dated approximately six months after symptoms had first become manifest.

On admission the right lid was pendulous and relatively immobile. A tumour was present which measured 4 cm. horizontally and 2 cm. vertically. The thickness of the lids measured with calipers at the time of admission, was 6 to 7·5 mm. The skin of the lids was not discoloured and was freely movable over the underlying tumour. The swelling conveyed the impression of consisting of one large soft smooth central mass with a smaller one on either side of it. These masses were freely movable over the tarsal plate. The palpebral fissure was very much constricted and the outer half of the upper lid appeared to overlap the lower lid. The apparent palpebral fissure measured 20 mm. × 3 mm. (Fig. 1).

The lid was everted with considerable difficulty. The palpebral conjunctiva was thick, lustreless, of a pinkish-grey colour and resembled somewhat sodden blotting paper. It was particularly thick and appeared in rolls in the neighbourhood of the accessory lacrimal gland. The tarsal plate was not involved and the neoplasm did not seem to have materially infiltrated or penetrated into the underlying sub-conjunctival areolar tissue. The swelling of the left lid presented practically identical features. It entirely overlapped the lower lid, the only aperture being formed by the forward thrust of the upper lid by the pressure of the swelling against the eye-ball. The tumour measured 4·5 cm. × 2·3 cm., while it registered 5 to 6·5 mm. in thickness. In each case, with the exception of a moderate degree of chemosis about the accessory

lacrimal glands, the globes appeared normal; the corneae were clear, the anterior chambers were of normal depth, the pupils were regular and active and the fundi were normal. Tension was not raised and vision with correction was 6/6 with each eye. For purposes of comparison, the lids of a patient of practically the same body weight, measured 2·7 mm. in thickness.

FIG. 1. January 8, 1938.

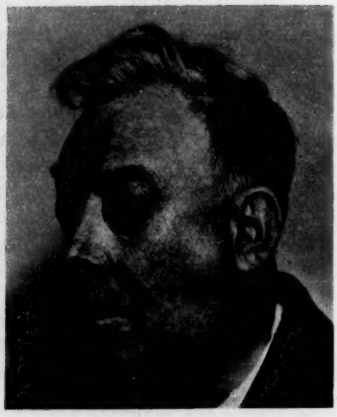

FIG. 2. April 19, 1938.

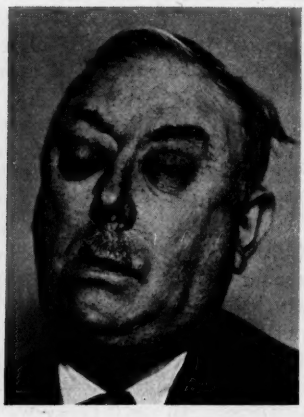

FIG. 3. April 28, 1938.

FIG. 4. May 25, 1938.

Glandular System.—There was a uniform adenopathy. The preauricular and the postauricular glands, the submaxillaries, submental, the anterior and posterior cervical glands were all enlarged, the preauricular being larger than the others. The

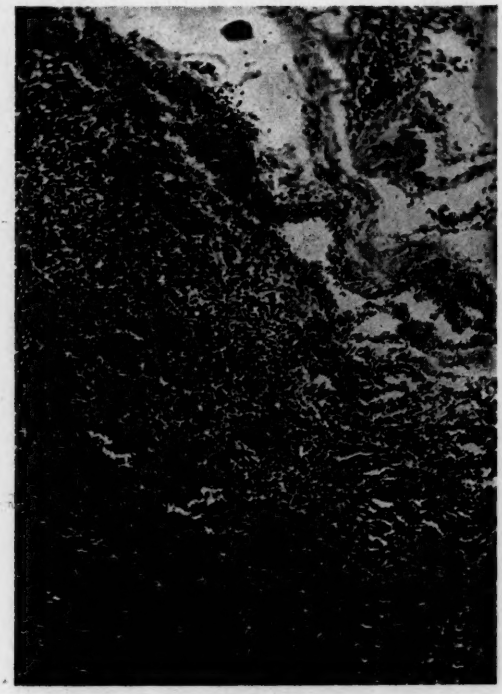

FIG. 5.

Paraffin section of tissue of left upper lid (low power). Densely packed lymphoid cells, medium in size.

axillary, the inguinal, and the epitrochlear glands were also enlarged. The heart was slightly hypertrophied to the left. B.P. registered 142/84. The respiratory system was normal.

Abdomen.—The patient was very obese; there was no tenderness or rigidity and no organs or masses were palpable owing to the adiposity. The hernial orifices (inguinal) were slightly enlarged.

Central Nervous System.—The superficial and deep reflexes were equal and active. Babinski's sign was negative; the cranial nerves were intact.

A film of the chest showed marked enlargement and increased density involving both lung roots. This enlargement of the roots of the lung might be due to hypertrophied glands adjacent to the hila and in the region of the tracheal bifurcation.

A biopsy was done on the left upper lid. A vertical section was made through the conjunctiva of the everted lid and through

FIG. 6.

Paraffin section of tissue of left upper lid (high power). Densely packed lymphoid cells, of medium size, exhibiting occasional mitotic figures. Corresponds to elements encountered in section through lymph gland.

which the sub-conjunctival tissue protruded. This tissue was similar in colour and appearance to the centre of a boiled potato and had the consistency of cream cheese on cutting. The edges of the conjunctival tissue were brought together with some difficulty owing to stretching brought about by the tumour. No complication followed convalescence.

A biopsy of one of the right posterior cervical glands was done. The tissue removed was larger than a peanut and very soft in consistency. One portion of the capsule was nicked and a thick cheesy-like material escaped, similar to that noted in the lid incision.

Paraffin sections of the lid showed densely packed lymphoid cells, rather medium in size, exhibiting occasional mitotic figures and corresponding to the elements met with in the lymph glands described below. (Figs. 5 and 6.) Such a proliferation would be classed amongst the more immature types of lymphatic

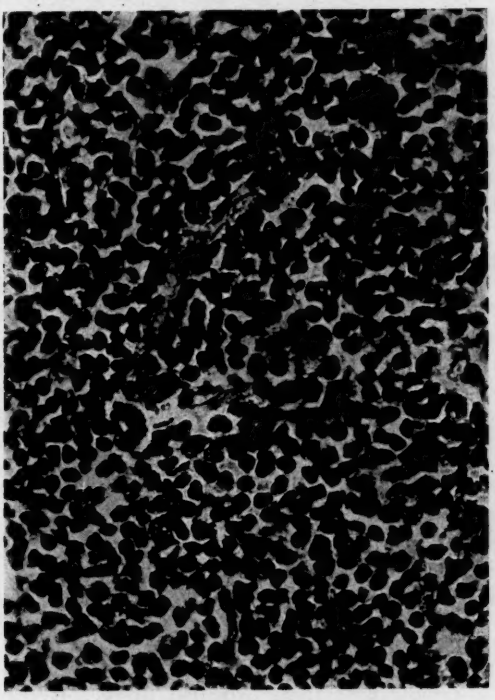

FIG. 7.

Paraffin section of tissue from posterior cervical lymph gland (high power). Loss of normal structure due to intense proliferation of closely packed, small, round lymphoid cells. The cells are quite uniform, a trifle angular in some areas, with a suggestion of clinging to the reticulum.

leukaemia with appreciation that it borders on a lymphosarcomatosis. The findings are very similar though somewhat more immature to that frequently described under the term "lymphatic leukaemia cutis."

Paraffin sections of the lymph gland showed a loss of normal structure due to an intense proliferation of closely packed, quite small, round lymphoid cells. The cells were quite uniform, they appeared to be a trifle angular in some areas, with a suggestion of clinging to the reticulum. The picture presented here again corresponds to that found on the borderline between a lymphatic leukaemia and a relatively mature form of lymphosarcomatosis. We were inclined from the general appearance to classify it amongst the rather immature forms of lymphatic leukaemia. (Fig. 7.)

Morphological blood examination—January 10, 1938.

R.b.c.:—2,700,000. W.b.c.:—5,600. Haemo.:—57 per cent. (8·7 gm/100 c.c.). C.I.:—1·06.

Red Blood Cells.—Stain well. Slight anisocytosis and no poikilocytosis. No polychromatophilia, punctate basophilia or nucleated reds. Reticulated reds:—less than 1 per cent. filament. Index of haemolysis:—460. Fragility:—normal. Corpuscle volume:—·28 per cent. Average corpuscle volume:—104 cubic microns. Haemoglobin concentration:—97. Majority corpuscle diameter:—7·8 microns. Sedimentation velocity:—(corrected for C.V.)=25, moderately increased.

White Blood Cells.—

Polys.	(Segment	rod)	Myel.	Eosin.	Mast.	Monos.	Lymphs.
25·5	(22·25	2·7)	4	0	·75	5·5	65·25 per cent.
1,428	(1,264	154)	56	0	42	308	3,766 per cmm.

There is a reduction in polys. with some immature forms. The lymphocytes are increased due to the presence of immature lymphoblastic elements.

Platelets:—100,000. Usual size and shape.

Bleeding time:—Not appreciably prolonged. Rumpel-Leeds: negative.

Coagulation:—fibrin 5 min. congealing 9 min. with calcium 7 min. retraction present.

Plasma.—Yellow with faint greenish tinge. Refraction:—71.

Van den Bergh:—Direct, prompt, negative; delayed, faintly positive indirect:—less than 0·3 units.

Conclusions.—The picture shows a quite marked anaemia with tendency for the cells to be somewhat larger than normal, and poor regenerative ability. There is a moderately increased sedimentation velocity. The white blood cells show deficiency in the number of the myeloid elements, with some immature forms. The

lymphocytes are increased, and this increase appears to be due to the presence of occasional lymphoblastic cells, corresponding to those met with in the lymph glands.

The blood picture is neither typical of a lymphatic leukaemia, nor lymphosarcomatosis, so we are inclined to believe that the condition represented here probably lies on the borderline between these two diseases. That such an intermediate type might well occur is understood by the intimate relationship between these two conditions and that the latter is simply of more immature undifferentiated nature. For the purpose of classification it is probably best considered an aleukaemic lymphosis, *i.e.*, an aleukaemic form of lymphatic leukaemia.

Morphological blood examination—January 27, 1938.

R.b.c. :—2,500,000. W.b.c. :—5,000. Haemo. :—51 per cent. (7·9 gm/100 c.c.). C.I. :—1·02.

Conclusions.—The blood picture is essentially the same as at previous examination (January 10, 1938). There is a slight general change for the worse.

Morphological blood examination—May 19, 1938.

R.b.c. :—1,700,000. W.b.c. :—8,000. Haemo. :—34 per cent. (5·3 gm/100 c.c.). C.I :—1.

Conclusions.—The general character of the blood picture met with at the last examination (January 27, 1938), is maintained. There is a general change for the worse, indicating in all probability a more extensive involvement of the haemopoietic system.

When treatment was inaugurated by X-rays each lid registered 13 mm. of swelling, the hyperplasia having very manifestly increased through delay. On April 19, 500 R. units filtered through 3 mm. Al. to the left eyelid, the cornea being protected by lead plate. On April 20, 250 R. units were applied to the same lid (Fig. 2). An April 27, 350 R. units were applied to the right eyelid, and on April 28, 400 R. units were applied to the same lid (Fig. 3). The total dosage to each lid was 750 R units. On May 25, after practically one month's delay, X-ray treatment was resumed and 125 R. units were applied to each lid. (To date—1,000 R. units have been applied to both lids.)

The effect of this treatment has been most spectacular in that the growths have apparently melted away (Fig. 4). The patient can now raise his lids and find his way about with ease, and the thickness of each lid registers 3 mm. Such improvement, however, unfortunately must only be regarded as local as the patient's general condition is definitely worse since he has come under our observation. The glands, particularly about the face, are distinctly more enlarged. His weight has decreased by nearly 50 pounds, which now affords one an opportunity of demonstrating the spleen to be rounded at its edge and to exend 8 cm. beyond

the costal margin, while the liver is hypertrophied and nodular and also extends 8 cm., beyond the rib margin. His breathing on the slightest exertion is laboured manifesting an additional hyperplasia of the mediastinal glands, while his haemoglobin has fallen from 57 per cent. to 34 per cent.

Discussion

If one turns to the ophthalmological literature on this subject, one is at once impressed by the fact that there exists considerable confusion because of a lack of uniformity in the terminology employed. Most authors emphasise the difficulties of precise diagnosis and classification.

Duke-Elder, in his textbook of Ophthalmology, Vol. 2, p. 1,796, 1938, places lymphomata or lymphoblastomata under tumours of the haemopoietic system. He states at the outset that a differentiation of tumours arising from the lymphocyte-forming tissues is difficult, and in the conjunctiva the difficulty is increased by their great variety. They may be divided into two classes depending on whether they are simple or malignant.

1. Simple lymphomata represent a variety of opinions: pathologically they manifest a marked hyperplasia of lymphoid tissue. Axenfeld (*Arch. f. Ophthal.*, Vol. XXXVII, p. 4, 102, 1891), and Meller (*Ibid.*, 62, 130, 1906), consider that they are always associated with some disease of the haemopoietic system; while other authorities are equally convinced that they occur without any constitutional disease—Coats (*Ann. of Ophthal.*, Vol. XLIV, p. 235, 1915). No adequate explanation has been brought forward to account for the appearance of the latter class. It has been suggested that they may arise from foetal remains in the orbit—Boerma (*Arch. f. Ophthal.*, Vol. IV., p. 219, 1894); Goldzieher (*Ibid.*, Vol. XLVII, p. 71, 1907).

A. Lymphomata occur with these diseases associated with a hyperplasia or metaplasia of the lymphoid and blood forming tissues and are frequently noted with enlargement of the lymph gland, spleen, and liver, with changes in the bone marrow. They may be manifest in cases of lymphatic leukaemia which they resemble in their occurrence and histologically the multiple tumour-like nodes seen in the skin in this disease—lymphatic leukaemia cutis. Less frequently they may be met with in Hodgkin's disease, in Banti's disease, in pseudo-leukaemia and other haemopoietic disturbances. Unlike the case under consideration they are small, hard, red fleshy tumours, painless and of slow growth. They may invade the palpebral and bulbar conjunctiva, the plica and limbus, and may also extend to the cornea. Note a case recently reported in the Journal of the Canadian

Medical Association, Vol. XXXVIII, No. 5, p. 477, by Aylesworth. Histologically the growth is made up of lymphocytes, the blood vessels being supported by a sparse and delicate connective-tissue framework. They may show a tendency to infiltrate and a differential diagnosis between a benign lymphoma and a malignant sarcoma may constitute a histological puzzle. As Duke-Elder continues to suggest, much truth may be gained from the blood picture.

B. Lymphomata occurring without evidence of constitutional disease present the same histological picture and clinical appearance as do those associated with leukaemia. They may be bilateral, symmetrical, and are met with most frequently in middle aged adults. There may be an associated enlargement of the lymph gland. These tumours may be of slow growth. Meyer (*Arch. f. Augenheilk*, Vol. LXXXIX, p. 156, 1921), has described one case which had persisted for seven years.

2. Lymphosarcomata, according to Duke-Elder, when a definite element of malignancy is evident, are rarely met with. Histologically, as I have attempted to point out, the comparison to a simple lymphoma is close; a mass of small round cells with some larger reticular cells. The tumours are fat and soft, of rapid growth, high local malignancy, with a special tendency to spread by the lymphatics.

R. Kummel (Lubarsch and Heinke Handbuch der spec. Path. Anat., Vol. XI, p. 2, 225), disposes of the subject more briefly. He concludes that in order to attempt to establish a definite diagnosis a blood picture is essential; even then he considers an exact diagnosis to be frequently a matter of doubt.

It would seem quite apparent, therefore, that in consideration of the lymphoid proliferations of this region, we must recognise that there is first of all a very rare type of either benign or malignant growth which is of purely local character, and is not associated with any generalised systemic disease of the other lymphoid tissues of the body. Nevertheless, by far the majority of the cases, in which lymphoid proliferation occurs in this region, are part and parcel of one of the various haemopoietic diseases, which involve as a rule, sooner or later, the bone marrow, spleen, lymph glands, and even other lymphoid tissues of the body.

For years, and in fact, since the original work of Hodgkin, authors interested in haematology, have been concerned with the identification and separation of these various processes into clear cut disease entities. More recently, however, there has been a definite trend to an appreciation that these various conditions form an inter-related series with no sharp lines of separation, but a gradual grading from one into the other. Waugh* has recently

* WAUGH, T. R. — The inter-relation of various systemic haematopoietic processes. *Amer. Jl. Med. Sci.*, Vol. CXCIII, p. 337, 1937.

emphasised this view in an aticle on the inter-relation of various systemic haemopoietic conditions, and from our standpoint it seems most important that we appreciate this trend as it explains the great difficulties which have arisen in the past in the sharp separation of these various conditions, and allows us to approach the subject from the much more natural viewpoint of placing any particular case in its proper position in the series, rather than trying to separate it sharply from other closely related conditions.

As regards treatment: X-rays were first employed by v. Duyse (*Arch. d'Ophtal.*, Vol. XXV, p. 402, 1905), and satisfactory results have been reported by Tiscornia (Rev. Assoc. Med. Argent., XXXVI, 818, 1923)—quoted by Duke-Elder and Speciale-Riccichi (*An. di Ottal.*, Vol. LV, p. 412, 1927). Radium has been used to advantage by Shannon and McAndrews (*Amer. Jl. Ophthal.*, Vol. XV, p. 821, 1932).

Turning to the particular case under discussion here, we find that it consists of one of the rarer, more active types of lymphoid proliferations which lie on the border-line between the more common forms of lymphatic leukaemia and the lymphosarcomatoses.

That this might be the case was first suspected from the character of the tissue removed from the eyelid. It showed a proliferation of lymphoid cells which, while comparatively uniform and closely packed together, were larger and distinctly less differentiated than those met with in the usual type of leukaemia cutis. Biopsy of the lymph gland showed a similar picture.

When the blood examination was first carried out this idea was substantiated again by the findings as there was present in the circulating blood a number of quite immature lymphoblastic elements. The total number of lymphoid cells was increased, but there was not the very high figures met with in the usual lymphatic leukaemia. This case, therefore, is one of a systemic lymphoid proliferation, which apparently from the progressive changes in the blood picture has involved the bone marrow, spleen, and lymph glands of the patient, as well as in an unusual manner, the eyelids. The proliferation is of a comparatively active form, and falls in its intensity and character between that met with in lymphatic leukaemia and that in lymphosarcomatosis. For this reason it is best classified as a leukaemic form of lymphosis, with unusual involvement of the eyelids and with an activity of cell proliferation bordering on the lymphosarcomatoses.

A NOTE ON CHRONIC IRIDO-CYCLITIS
With Special Reference to the Sarcoidosis of Boeck

BY

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THIS condition is not one with which the general physician makes much contact, and without specialised ophthalmological knowledge he needs to walk somewhat carefully. But it is a disease of paramount importance to the ophthalmologist and his patient by reason of the obscurity of its aetiology and because treatment is often unable to prevent serious damage to the sight. It should be understood that my remarks are confined to chronic iritis which has either been insidious from the outset or starting with an acute attack has failed to clear up in the usual way. As regards pathogenesis the literature is rarely helpful and often misleading. Syphilis, tuberculosis and focal sepsis are the three main factors indicated in causation but their relative importance is much disputed. As a result of an investigation at the Manchester Royal Eye Hospital Gray Clegg found that in 55 cases the cause was syphilis in 6, tuberculosis in 2, a septic focus was found in 12, whereas the cause was quite unknown in 35 (63·6 per cent.).

To take tuberculosis first it is thought to be the basis of chronic irido-cyclitis in a proportion varying from 5 to 50 per cent. according to the views of individual writers—Doggart (1936). An idea of the grounds, or lack of grounds on which the diagnosis may be based can be obtained from a paper by Irons (1931) and the discussion which followed it. It seems that the diagnostic criteria vary somewhat. The absolute diagnosis is established when tubercle bacilli are found on section. Irrefutable clinical evidence is the appearance of tubercles—though this is very rare. When the condition responds to tuberculin treatment it may be presumed that it has been specific. But the presence of healed, latent or sub-clinical tuberculous lesions or a positive tuberculin reaction should, in my view, not be allowed to establish the diagnosis. This type of iritis is very rarely associated with an active lesion—with the exception of miliary spread—so that when strict proof is demanded it will be seen that tuberculosis plays a small part in aetiology. Syphilis undoubtedly is responsible for a few cases—acute, chronic or Herxhiemer reaction. The American figures are in the neighbourhood of 18 per cent., but it must be remembered speaking approximately that while in America 20 per cent. of the coloured and 10 per cent. of the white population

are syphilitic; in England the corresponding figure is nearer 3 per cent. There is a good deal of difference of opinion as to focal sepsis as a cause of chronic irido-cyclitis though in the acute disease it may be a considerable factor—Copps and McCormick (1937). Doggart's criticism that the thesis has yet to be proved is in keeping with experience in general medicine. Irons and Brown (1923) are prepared to admit that though focal sepsis may be found it may not be causal. And indeed there may be little difference in the incidence of septic foci in these patients and apparently healthy people. Certainly heroic investigation and eradication has quite failed to solve the question of chronic iritis. Doggart mentions the rare condition of heterochromia iridis and the frequency of iritis in menopausal women regarding this as propably endocrine in nature. It is of course a classical observation that herpes zoster ophthalmicus is a cause of subacute iritis. In addition iritis is rarely but irrefutably seen in the following illnesses — typhoid, influenza, malaria, leprosy, brucellosis, diabetes, gout and lastly the association with gonorrhoea is well known. In most of these latter conditions the iritis is likely to be acute rather than chronic.

To this list there is now to be added a new type of chronic iritis, that of the sarcoidosis of Boeck. Briefly this is a disease of an unknown pathogenesis in which sarcoid granulation tissue is deposited in nearly every organ in the body. On section this resembles the epithelioid reaction of hyperplastic tuberculosis but the clinical sequence of these benign deposits builds up a composite picture from which an accurate diagnosis can be made. A good clinical description is to be found in the paper by Scott (1938). The uveal tract is almost invariably, at an earlier or later stage, the seat of these deposits and a chronic inflammatory reaction results. Such a case is well described by Gordon (1937). A woman gave a story of six years' ocular inflammation. There were sarcoid skin lesions (biopsy), lung infiltrations, a palpable spleen and a negative Wassermann reaction. I have seen two similar cases. The first, a young man of twenty-five was found on routine insurance examination to be almost blind in the right eye due to chronic irido-cyclitis. The other eye was also slightly affected. On being overhauled it was found that he had considerable mediastinal and hilar gland enlargement. All further investigations were unhelpful and his general health was good. The glands settled down over a period of several months but the iritis continued to show slow progress. The second patient had been diagnosed as having a chronic sarcoid skin patch and when I came to examine her I found that she had a slow and quite asymptomatic irido-cyclitis. In both instances there was no gross inflammation, very little keratitis punctata and the posterior

synechiae were brown and shaggy. These points together with a painless, insidious and relatively benign course may mark it off from other forms of uveitis. It has been suggested that uveo-parotid fever may well be a clinical mutation of sarcoidosis though it must be remembered that in some fatal cases miliary tuberculosis has been shown. Krumskal and Levitt (1935) described a case of uveo-parotid fever in which only sarcoidosis could account for all the facts. A woman of 34 showed the positive findings of chronic irido-cyclitis, firm parotid swellings, chronic skin lesions on the legs, an isolated gland in the neck, large glands at the roots of the lungs, an enlarged spleen, pyrexia and negative tuberculin reactions. Section of the gland in the neck showed a hyperplastic epithelioid reaction with giant-celled systems but no caseation and no tubercle bacilli. The authors regard this case as evidence for the tuberculous nature of Heerfordt's syndrome but in future I think that we will have to think of sarcoidosis.

With this kind of case in mind Professor Cohen asked me to see to what extent patients with chronic uveitis were actually suffering from generalised sarcoidosis. It was possible to examine twenty-five cases, all of whom were very chronic and intractable and in whom no cause had been found. No acute cases were included in this group.

Analysis of the Clinical Material

The patients who form the material for this paper were seen at St. Paul's Eye Hospital, where I was able to go into their history carefully and examine them. They had been in most cases attending the out-patient department for several years. Most had been investigated in hospital for focal sepsis, tuberculin reactions. Wassermann reactions, protein sensitivity, and so on. In addition it was possible to have their chests radiographed for the exclusion of active tuberculosis and sarcoid infiltrations of the glands and lung fields. The details of my findings are in the accompanying tables. Grouping according to age of onset and sex seems to be justified by the closeness of the ages with only one exception in Group II. Though the numbers are small I think that this exact grouping must have some aetiological significance. The average age in Group I is 21, Group II is 38, and in Group III is 50.

The general health before and during the illness was uniformly good. In Group III one began to find the chronic degenerative changes so common at that age—obesity, villous arthritis and some hyperpiesia. There was no true rheumatoid arthritis in any patient. In a detailed survey there was no outstanding family ailment. And though these 25 patients subtended a large number of relatives there were no other instances of iritis. The evidence

GROUP I—YOUNG WOMEN

No.	Age	Duration	Onset	Course	Extent	Vision	Associated Lesions
1	25	5 years	Slow	Chronic	Bilat.	Reads	Slight mediast. glands
2	25	5 years	Acute	Recurr.	Bilat.	P.L.	Psoriasis
3	30	14 years	Slow	Recurr.	Bilat.	P.L.	Nil
4	25	5 years	Slow	Chronic	Bilat.	Reads	Mitral stenosis
5	26	2 years	Insid.	Chronic	Bilat.	P.L.	Cervical adenitis
6	25	2 years	Acute	Chronic	Bilat.	Nil	Nil
7	16	9 years	Acute	Chronic	Bilat.	P.L.	B.P.=170/110
8	52	20 years	Acute	Recurr.	Bilat.	Nil	Glossitis
9	55	37 years	Acute	Recurr.	Bilat.	Nil	Poor physique

GROUP II—MIDDLE-AGED MEN

No.	Age	Duration	Onset	Course	Extent	Vision	Associated Lesions
10	40	7 years	Insid.	Chronic	Bilat.	Reads	Dyspepsia—no ulcer
11	53	6 years	Acute	Recurr.	Bilat.	Reads	Healed acute nephritis
12	45	6 years	Acute	Recurr.	Bilat.	Reads	Nil
13	42	1 year	Grad.	Chronic	Bilat.	Reads	Nil
14	36	10 years	Acute	Recurr.	Bilat.	Good	3 small lipomata
15	45	2 years	Grad.	Chronic	Unilat.	Nil R.E	Nil

GROUP III—ELDERLY WOMEN

No.	Age	Duration	Onset	Course	Extent	Vision	Associated Lesions
16	52	2 years	Grad.	Recurr.	Bilat.	P L.	Nil
17	55	1 year	Acute	Recurr.	Unilat.	Normal	Villous arthritis knees
18	54	10 years	Grad.	Chronic	Bilat.	Reads	Radiol. phthisis B.P. 170/110
19	52	3 years	Grad.	Insid.	Bilat.	Reads	Very slight mediast. glands
20	62	5 years	Acute	Slow	Bilat.	P.L.	General health good
21	37	2 years	Insid.	Slow	Bilat.	P.L.	Nodule thyroid
22	57	4 years	Acute	Recov.	Bilat.	Fair	Vision improving
23	56	8 years	Acute	Recurr.	Bilat.	Reads	B.P. 175/120
24	55	1 year	Acute	Recurr.	Bilat.	Reads	Healthy
25	58	1 year	Insid.	Chronic	Bilat.	P.L.	Nil

in favour of a tuberculous aetiology was scanty. Two relatives had died of phthisis—this figure being rather less than one would have expected in any similar group of families. Two brothers of one patient were in a children's sanatorium with suspected but not active abdominal tuberculosis. One girl of Group I had had glands removed from her neck four years previously and they had been regarded on macroscopic examination as tuberculous. There were two firm glands remaining which clinically might have been due to tuberculosis or sarcoidosis. Biopsy was refused. One woman of Group III with a long history of chest symptoms had a good deal of radiological thickening of the lung fields which was thought by the radiologist to be tuberculous. The sputum was not, however, positive. I have ignored the results of the tuberculin tests though it should be noted that in sarcoidosis it is said that the reactions are more frequently negative than in the normal population.

At the time that I saw these patients the illness had been present for many years—in one instance for 37 years. In Group I the average duration was 11 years. In Group II it was 5 years and in Group III approximately 4 years. There seemed undoubtedly to be two distinct methods of progress but whether this meant more than one pathogenesis it was not possible to say. On the one hand there was an initial acute uniocular iritis settling down into a chronic irido-cyclitis. This was followed at a varying period by recurrent attacks in which the other eye was also involved. On the other hand in rather fewer cases the onset was insidious and painless. Sometimes there was no indication of the disease process until a casual covering of the sounder eye revealed the impairment in the other. Deterioration then took place relentlessly over a matter of years. Both these types were equally represented in all three groups of patients.

When one came to examination good general health was found and no symptoms beyond slight pain and failing vision. As might be expected this gradual failure in adults was accompanied by a fair amount of depression and lack of interest with relative failure of adaptation to the new conditions of limited sight. In addition to the general physical overhaul a special search was made for sarcoid lesions, such as skin changes, adenopathy, splenomegaly, and phalangeal deformities together with radiography of the lungs. There were very few positive findings. The cervical adenitis and the radiological phthisis have been mentioned. Two patients had slight mediastinal gland enlargement. One man had three small subcutaneous nodules clinically resembling lipomata. One of these was excised and on section showed that this was the case.

As for treatment these patients had had every conceivable

measure during the years that they had been attending the hospital. In the first place any focal sepsis found had been eliminated. As well as local mydriatic measures, they had had courses of organic arsenicals, tuberculin, gold preparation, and autogenous and stock vaccines. Together with mild fever therapy and protein shock with T.A.B., milk injections, autohaemotherapy, peptone, and so on. Several patients had had to sacrifice an eye and many had submitted to iridectomy. In passing, the most optimistic therapeutic report in the literature is the electric pyrotherapy reported by Whitney (1935). But despite the best of treatment and supervision the prognosis as shown by this series is poor. Though it must be remembered that I saw no patients who could have been regarded as responding to therapy or making a spontaneous cure. In all but two patients both eyes were affected. And in only one patient was the vision improving. The eyesight of the rest—as I have briefly indicated in the tables—was greatly limited. The least affected were still able to read, others were deprived of their reading but could grope their way about, but three patients were quite blind. From the grouping it is clear that the worst results are seen in Group I. Whereas in Group III process was slower and it was still possible for instance to continue with household duties.

Such was the material that I was able to examine and though the results were not strikingly positive it was possible to draw the following conclusions.

1. The incidence of non-specific chronic irido-cyclitis seems to fall into three age/sex groupings.

2. Apart from this, present day clinical methods are unable to link the illness with any of the known aetiological causes. It may therefore be a disease *sui generis*.

3. It follows a chronic progressive course, is not amenable to treatment and the visual prognosis is poor.

4. Sarcoidosis is not a cause in the majority of instances.

Acknowledgment. I would like to thank the honorary staff of St. Paul's Hospital for their courtesy and co-operation.

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A STUDY OF DIET IN RELATION TO HEALTH. DARK ADAPTATION AS AN INDEX OF ADEQUATE VITAMIN A INTAKE*

II.—A New Photometer for Measuring Rate of Dark Adaptation

BY

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IN the first paper of this series (Mutch and Griffith, 1937) the results were reported of a preliminary study in which a modification of Edmund and Clemmesen's method (1936) was used to assess degrees of dark adaptation by measuring the power of distinction with a series of test letters ranging from black to near white over a wide light range. The test was incorporated in the scheme of an extensive clinical and dietary survey, in order that its usefulness for diagnosing vitamin A deficiency might be judged. However, it soon became apparent to the field worker in charge of this test that the results were difficult to interpret in many cases owing to the presence of two variables (*i.e.*, the light control by means of Tscherning glasses, and the range of test letters). The power of distinction might be excellent at the low illuminations and poor at high illuminations or vice versa. For this reason a new adaptometer was designed, using a black test letter only and with another type of light control which could measure finer gradations than the Tscherning glasses. The new apparatus had the further advantage that the principles involved were the same as in the other apparatuses designed for the same purpose, *i.e.*, measuring a light threshold only, and not involving any estimation of power of distinction between greys.

The Apparatus

General Construction.—The instrument† is built within a long narrow wooden box, 100 cm. long, 20 cm. broad, and 20 cm. high, supported on a tilting framework so that it can be set at any angle convenient to the observer, who looks into it as D (Fig. 1). It

* This work was done in connection with the Carnegie United Kingdom Trust dietary survey.

† In the description of survey results to follow in the next paper of this series, the apparatus will be referred to as the Rowett adaptometer.

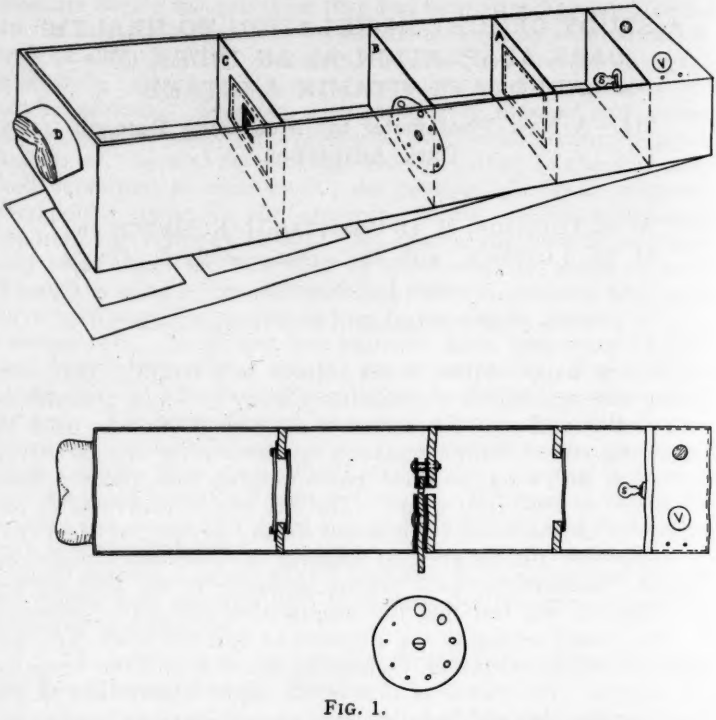


FIG. 1.

is divided by partitions into four compartments, as indicated in the figure, the first of which contains the light source S, and has its walls painted matt white; the other compartments have dark stained walls. A hole 8 cm. square, cut in partition A, is covered with a rather dense opal glass (Chance's Pot opal), and a similar hole in partition C is covered with a thinner plate of opal glass (Chance's single flashed opal). The compartment to the left of C is a viewing chamber, the subject looking into it with both eyes towards the illuminated surface of C, resting his head against a metal visor fitted into the end of the box at D. The illumination of the opal surface C can be varied in steps by turning a wheel diaphragm inserted in front of a circular aperture in screen B; the diaphragm has nine circular holes of graded diameter, any one of which can be brought on to the axis of the instrument and held there by a spring and ball catch, the hole being indicated to the operator by a number engraved on the exposed edge of the disc. Aperture 9, the largest, is also indicated by a notch which can be

felt in the dark, and the progressively smaller apertures follow in descending numerical order as the disc is rotated.

The holes were designed to range in diameter from 1·20 cm. to 0·075 cm., giving a range of illumination of surface C in proportion to their areas, that is, of 256 : 1, the illumination being halved by each change from one hole to the next smaller. It was not considered worth while to have special tools made for drilling the apertures and their diameters were not exactly those specified to the mechanic. The actual diameters and corresponding illuminations of the screen C are given in Table I.

TABLE I

APERTURE	DIAMETER cm.	ILLUMINATION Micro-lux	Milli-ft.-cndls.
9	1·13	2790	0·260
8	0·795	1390	0·129
7	0·570	710	0·066
6	0·420	388	0·036
5	0·300	225	0·021
4	0·205	92	0·0086
3	0·132	44	0·0041
2	0·096	23	0·0021
1	0·059	8·7	0·00081

Test Object.—The test object used during the course of this work was a letter E of dimensions as in Fig. 2, cut from black paper and mounted between two lantern slide cover plates. It can be dropped into a slot so as to lie about half an inch in front of the opal glass C on the observer's side and it can be withdrawn by the operator by means of a wire bridle, turned and reinserted easily and quickly, even in complete darkness. The subject sees the test object against the feebly lighted surface C, the brightness of which is controlled by the diaphragm setting. He knows he must look for the form E, but does not know in which direction the E has been turned when inserted by the operator.

The E test object was adopted as it had been found satisfactory in the method of Mutch and Griffith. After the survey work (to be described in the next paper) was completed, we compared the curves obtained with the E test object with those obtained using one which was thought might have certain advantages over the E. This consisted of a rectangle 5 cm. x 3 cm. cut out of black paper and mounted between two lantern slide covers as shown in

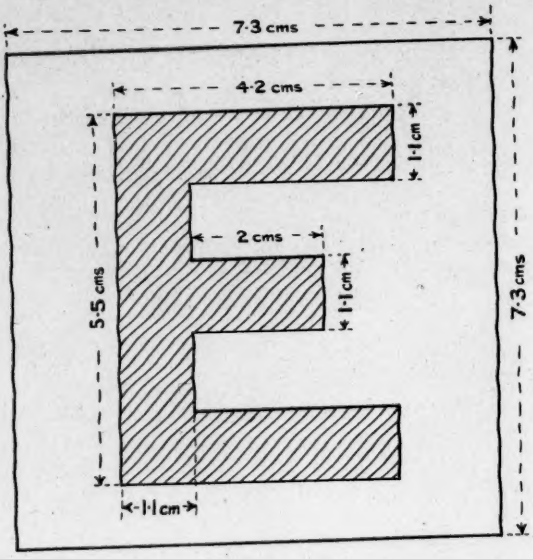


FIG. 2.

Area of Square - - -	= 53·29 sq. cm.
Area of E - - - -	= 15·07 sq. cm.
Light passing area - -	= 38·22 sq. cm.

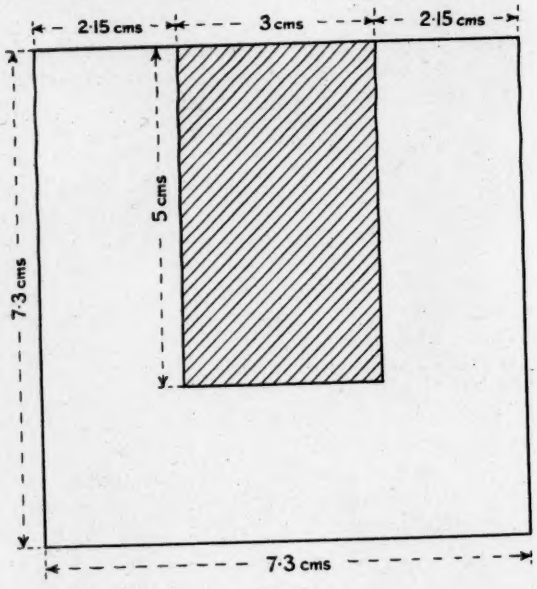


FIG. 3.

Fig. 3. The light-passing area of the slide is the same as that with the E. In practice, the subject is told to look for a square U of light. Comparative tests with these two test objects will be described later in this paper.

Light Source.—The source of light is a 6 volt 3 watt motor side-lamp bulb, operated at 5·00 volts from a 6 volt car battery. A good quality moving-coil voltmeter is permanently connected across the bulb and the voltage set to 5·00 volts by the use of a variable resistance. Occasional inspection of the meter during the course of a run is adequate to check electrical constancy so as to hold the illumination constant to ± 3 per cent. Control measurements made of the decrease in lumens output from several such lamps under operating conditions showed a drop of about 20 per cent. during the first 100 hours of use, after which the output decreased much more slowly, falling about 6 per cent. in the next 200 hours of use. The lamps used for routine work were therefore " aged " for 100 hours before being inserted in the instrument and were discarded after a further 200 hours of use. In order to secure initial uniformity, several dozen lamps were obtained from the manufacturers, and by photometric tests those individual lamps were selected which had a lumen output within ± 5 per cent. of the average. This control is essential, since the manufacturers' tolerance for lumens output for this class of lamp is stated as ± 15 per cent. and is therefore too wide for the purpose of the test. No significant differences were found in the response of the selected lamps to the ageing process, nor was trouble experienced from uncertain contact in lamp holders. The method described above for the control of the light source was adopted in preference to any method involving the use of a photo-cell because the control meter, a robust voltmeter, could be trusted to withstand a degree of rough handling which an adequately sensitive photo-electric measuring system would not endure.

Photometry of the Test Surface.—In order to ensure similarity between the instruments used in the routine tests and to make the results comparable with those of other observers, it was necessary to know the absolute brightness of the test surface. A rough estimate based on the measured intensity of the illumination of surface A (114 lux), on the geometry of the apparatus, and on the transmission factors of the opal glasses, indicated that the illumination of the test surface when aperture 3 of the diaphragm is in use must be in the order of 10^{-3} lux. A photographic method of photometry was chosen for the final evaluation. The standard source was an electric bulb calibrated against a Vernon Harcourt pentane lamp in the Physics laboratory of the University of Aberdeen, set up with suitable diaphragms at 410 cm. from a reference surface on which an Ilford Hypersensitive Panchromatic

plate could be exposed by withdrawal of the shutter of a special cassette. The cassette permitted light to reach a strip only of the plate and in front of this strip was an optical wedge of density rising in steps of 0·15 to 8·83. After exposure in this way a second strip of the plate was exposed in the same cassette for the same time in the plane C of the adaptometer, but without the optical wedge. On development, the plate therefore showed two blackened strips side by side, one evenly blackened by exposure in plane C, the other with blackening increasing in steps from exposure to the standard source, behind the optical wedge. By comparison of the blackening of the two strips in a photo-electric densitometer, the density of the wedge step requisite to reduce the calibration density to equality with that at plane C could be found, and hence the absolute value of the intensity at C was determinable. The values obtained are given in Table I. The illumination is not quite uniform over the whole of the test object: at aperture 3, for instance, it ranges from 44 microlux at the centre to 32 microlux at the corner where it is least.

When full allowance is made for variation of lamps and for electrical fluctuation, it is certain that the illumination at the centre of the test surface does not vary by more than ± 10 per cent. from the average, either during a run or on separate runs. Since the " Weber-Fechner fraction " at intensities of the order of 10^{-3} lux is not less than 50 per cent. (Hecht, 1924), this degree of variation in our apparatus cannot be significant; therefore elaboration designed to secure closer control of illumination is not justified. In support of this contention it may be stated that no advantage is obtained by decreasing the " step " of the diaphragm. Trials made in which the illumination was decreased in steps of 50 per cent. showed that such a step scarcely exceeded the observer's limit of discrimination of brightness. This would be expected from Hecht's results.

Technique

The instrument was originally used to determine the smallest aperture at which the E could be distinguished after fifteen minutes' preliminary dark adaptation, five minutes thereafter being allowed for the test. However, it became apparent that if the subject was left in the dark for longer than 15 minutes, he could, in many cases, obtain a lower reading. Hence preliminary experiments were started with time curves, and these not only confirmed the suggestion but indicated that the threshold at twenty minutes was greatly affected by the lighting to which the subject had been exposed before he entered the dark room. This is

explained by Graph I, showing curves* on the same subject after exposure to strong sunlight, and after being in a badly illuminated room.

Further work was therefore directed towards obtaining a method whereby this effect of antecedent light could be eliminated, as in this stage it was obvious that one subject could give a slow or rapid curve, depending on his previous environment.

The following technique was found to be satisfactory.

The subject is dark adapted until, at aperture 2, he can say which way the E is pointing. As will be shown later, this represents practically complete dark adaptation. The retina is then " bleached " on the apparatus now to be described, after which the test proper commences.

The bleaching apparatus is a modification of that used by Stargardt (1937), and consists of a metal bowl 2 feet in diameter and 18 inches deep, lined with plaster of Paris. The subject's head is supported on a chin rest on the lower edge of the bowl. Above his head is a 150 watt " pearl " electric globe, in the circuit of which a variable resistance is incorporated to control the effect of mains fluctuations. The subject is made ready in darkness with his chin on the rest, immediately following the preliminary dark adaptation and is warned that a bright light is going to be switched on. He is told to blink rapidly until the initial dazzle has passed off and then is required to keep his eyes open. Bleaching is timed by stop watch to take place for 4 minutes, after which the subject returns to the adaptometer for the test.

During the bleaching period, the operator should see that the subject does not close his eyes. With this type of bleaching apparatus, it is easy to keep the subject under observation.

The intensity of illumination of the surface of the bleaching bowl, measured with a visually corrected photo-electric cell, had a value of 93 foot candles at the point directly opposite the eye position, diminishing to 88 foot candles 60° from the centre. The intensity of the light and duration of exposure for bleaching were chosen by trial, firstly giving a rate of adaptation slow enough to be determined reasonably accurately, and secondly not so strong that undue discomfort is experienced by the subject.

A resistance for controlling mains fluctuations is essential. At the Rowett Institute the mains voltage is 230, and a recording voltmeter showed a daily maximum variation of ± 15 per cent. The lamp in the bleaching apparatus is controlled to run at about 200 volts. Constancy of illumination is ensured by frequent

* Since the illumination is increased by doubling at each step, the aperture numbers are the logarithms to the base 2 of the illumination, that at aperture 1 being unity. The curves are therefore logarithmic plots.

checks with an Ilford photo-electric meter, which was found adequately sensitive for the purpose.

In order to ascertain what effect uncontrolled mains fluctuations would have on the subsequent dark adaptation curve, six trained subjects were tested with the bleaching light at 180 volts and again at 220 volts. Table II shows the averages of the curves obtained. It will be seen that there is a significant difference between them.

TABLE II

Effect on Curve of Voltage Fluctuations
(Averages of 6 Curves)

Aperture	9	8	7	6	5	4	3	2
	M. S.	M. S.	M. S.	M. S.	M. S.	M. S.	M. S.	M. S.
Bleaching lamp at 180 volts - -	0 51	1 29	2 16	3 22	4 30	6 15	8 30	12 30
Bleaching lamp at 220 volts - -	2 9	2 47	4 8	4 53	6 47	8 32	10 51	14 16

After the bleaching the test proper begins. The adaptometer light is switched on, the diaphragm is set at aperture 9, and the subject looks into the eyepiece. In order to encourage the use of peripheral vision, he is told to look to one side. As dark adaptation proceeds, the subject shortly sees the illuminated square, and says in which direction the arms of the E are pointing. The time from the commencement of dark adaptation is taken by stop watch and recorded by one observer. Another observer rotates the diaphragm to aperture 8, and changes the direction of the E. The same procedure is followed down the scale to aperture 2.

It is found that some subjects, especially children, guess the direction of the E before they see it, and so give wrong responses. This is understandable, as an illumination approaching the threshold of light perception has to be looked for in each case. Hence from apertures 7 to 3 inclusive, two consecutive correct responses and at aperture 2 three consecutive correct responses, or a series of two correct, one wrong and two more correct responses are required. The time of the first correct response of the series is taken as the " time taken to aperture 2."

Curves obtained using the full technique as described above will be referred to as " standard curves."

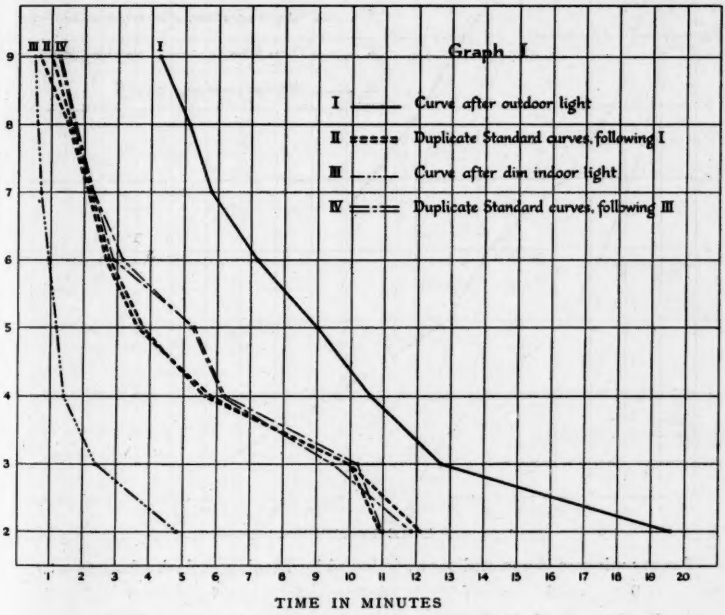
Critique of Method

(1) *Effect of previous Light on subsequent Dark Adaptation.*— It is necessary to amplify our comments on the effect of previous light on the preliminary dark adaptation in order to justify this somewhat tedious technique.

It will be seen from Graph 1 that the curves recorded for one person after varying previous lighting conditions are not parallel; the time taken from aperture 9 to 2 is longer when the previous light is stronger. Thus a simple assessment of this time does not give constant results. Furthermore, a preliminary dark adaptation of ten minutes followed by bleaching is inadequate to eliminate the effect of previous light. This is shown in Graph 2 where the subject was previously in strong outdoor light. A curve after 10 minutes' dark adaptation followed by bleaching (No. 2) and a standard curve (No. 3) are shown. This point was checked in two other subjects and similar results were obtained. The subject

D. McE., aged 10 years.

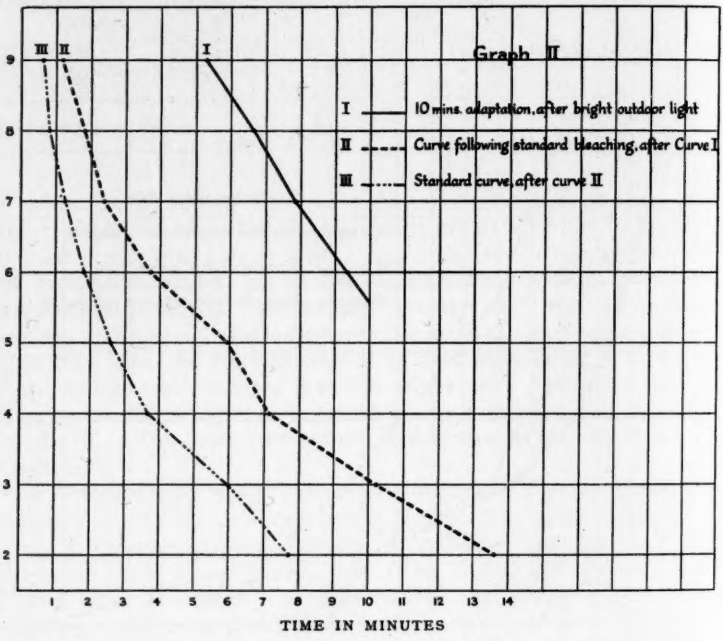
Curves I, II - 9/6/38.
Curves III, IV - 10/6/38.


Graph I
I Curve after outdoor light
II Duplicate Standard curves, following I
III Curve after dim indoor light
IV Duplicate Standard curves, following III
TIME IN MINUTES

Aperture	Curve I		Curve II		Curve III		Curve IV	
	M.	S.	M.	S.	M.	S.	M.	S.
9	4	15	1	0	0	37	0	30	1	15	1	15
8	5	7	1	38	1	37	0	35	1	40	1	40
7	5	43	2	9	2	13	0	45	2	15	2	14
6	7	10	2	50	2	43	0	59	2	54	3	10
5	9	0	3	37	3	45	1	10	5	20	5	18
4	10	35	5	52	5	40	1	25	6	10	6	10
3	12	45	10	0	10	0	2	23	10	12	9	30
2	19	35	12	5	11	0	4	50	10	58	11	55

whose curve is illustrated has consistently given, over several tests, some of the most rapid curves we have timed, and so might be expected to recover from the effects of strong illumination more quickly than the average. It appears, therefore, that complete dark adaptation is required in order to eliminate the effect of antecedent light. In further support of this contention it may be pointed out that Matthey (1933) has demonstrated that full preliminary dark adaptation is required in order to ensure a standard curve independent of previous light.

A. J., aged 18 years. 6/1/39.


Graph II
I 10 mins. adaptation, after bright outdoor light
II Curve following standard bleaching, after Curve I
III Standard curve, after curve II
TIME IN MINUTES

Twenty minutes in sun and snow, followed by 10 minutes dark adaptation:

9	8	7	6	5	4	3	2
M. S.	M. S.	M. S.	M. S.				
5 23	6 40	7 53	9 21	...	...	...	...

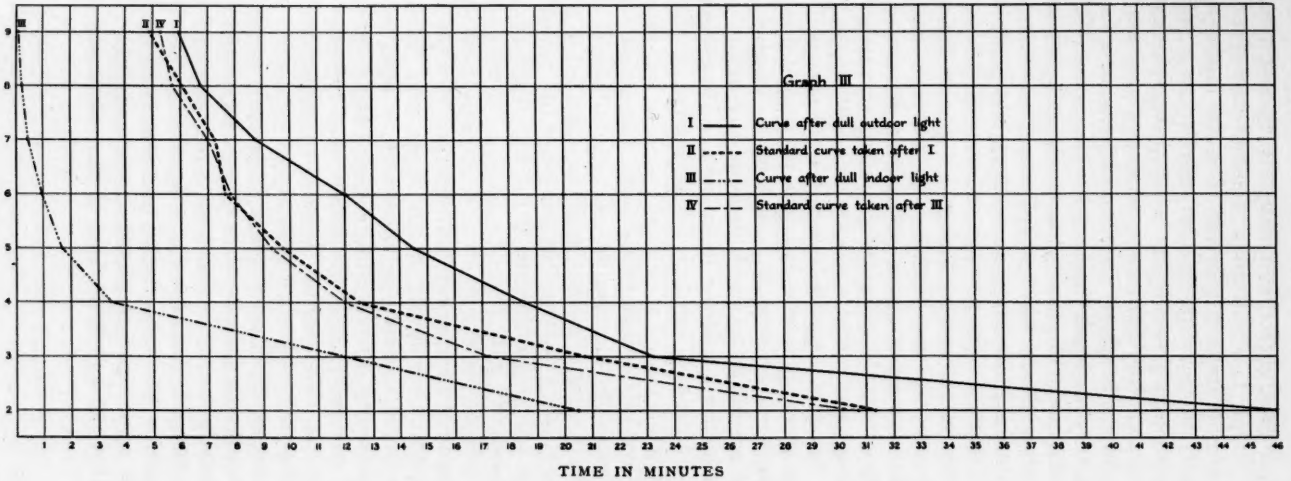
Thereafter, 4 minutes standard bleaching:

M. S.	M. S.	M. S.	M. S.	M. S.	M. S.	M. S.	M. S.
1 10	1 52	2 20	3 46	5 55	7 06	10 24	13 37

Four minutes standard bleaching on completion of above:

M. S.	M. S.	M. S.	M. S.	M. S.	M. S.	M. S.	M. S.
0 38	0 50	1 16	1 52	2 35	3 42	6 00	7 46

I. M., aged 10 years. 26/1/39.


Graph III
I —— Curve after dull outdoor light
II ----- Standard curve taken after I
III —·—· Curve after dull indoor light
IV — — — Standard curve taken after III
TIME IN MINUTES

Aperture	Curve I	Curve II	Curve III	Curve IV
	M. S.	M. S.	M. S.	M. S.
9	5 58	4 56	0 3	5 15
8	6 47	6 3	0 10	5 42
7	8 45	7 15	0 25	7 2
6	12 0	7 41	0 57	7 52
5	14 30	9 47	1 39	9 21
4	18 32	12 30	3 25	12 5
3	23 17	20 43	11 58	17 15
2	45 56	31 20	20 27	30 47

That our technique does, in fact, eliminate this effect of previous light is shown by Graph 1, which gives standard curves on one subject taken after strong outdoor light and after dim indoor light. Graph 3 shows that the technique is also effective in a subject whose curve is very much slower. The method was checked in this way on 10 children, with 32 pairs of preliminary and standard curves, and we are satisfied that the standard curve is independent of previous light conditions in all cases.

We consider this a point of great importance, and it is likely that the results of all methods, which adapt only for a short fixed period before experimental bleaching, must fluctuate considerably, depending on conditions of light previous to the test.

Another fact which was observed during this part of the work may be mentioned here. As the weather in the north of Scotland is not often brilliant enough to cause sufficient deflection of the dark adaptation curve for the purpose of testing our technique adequately, an attempt was made to use an artificial light source, consisting of four 200 watt lamps reflected at a distance of 2 feet from a whitewashed sheet. The interesting fact emerged that this brilliant artificial light, which was uncomfortable to look at from close quarters, had not nearly as much deflecting power as weak winter light (not sunshine), reflected from snow. There appears to be a qualitative difference in lights as far as retardation of dark adaptation is concerned.

(2) *The Visual Threshold and Impaired Dark Adaptation.*—It is of interest to compare the illumination of the test object with those at which clinical night blindness might be expected to make itself felt. Lythgoe (1938) gives the illumination of full moonlight as 1/100 foot-candle. The illumination of the test object at aperture 9 is therefore roughly 1/40 that of a surface illuminated by full moonlight. Lythgoe also states that the threshold of vision is 0·001 millifoot candle. Comparing this with the illumination of the test object at aperture 2 (allowing about 20 per cent. reduction of illumination of screen C owing to absorption of light by the slide), it will be seen that the resulting figure (0·0016 millifoot candle) is about 50 per cent. above the threshold of vision of the fully dark adapted eye. At aperture 1 the illumination will be about 30 per cent. below Lythgoe's threshold.

In the range between apertures 9 and 2, therefore, we should expect to find evidence of impaired power of dark adaptation.

With regard to the criteria by which impaired power of dark adaptation may be judged, two views have been held: (*a*) that it is the rate of adaptation which is affected and that everyone ultimately reaches the same light threshold when completely dark adapted, and (*b*) that the light threshold of the completely dark

adapted eye is changed. Tansley (1939) considers that night blindness of both types exists.

The former view, as interpreted in practice, assumes significance for all readings after bleaching. In the Birch-Hirschfeld and biophotometer methods, judgment is made on one or more of a series of readings taken during the 10 minutes following exposure to the experimental bleaching light. Corlette *et al.* (1938), using the biophotometer, base their diagnosis of normality solely on the readings taken at 20 seconds. Tansley (1939) points out the danger of this proceeding and our data support her warning. In certain cases (*e.g.*, those shown in Graphs 1 and 3) there are consistent differences, between a subject adapting rapidly and one adapting slowly, at each illumination throughout the standard curves. But in many cases, those who adapted slowly showed the delay only at the end, at apertures 3 and 2, and were within the normal range to aperture 4.

Wald, Jeghers and Arminio (1938), on the other hand, consider that adaptation is complete in from 30 to 45 minutes and that judgment should be based on the light threshold at that time. When their subject was on a diet of low vitamin A content, they found the threshold " at complete dark adaptation " to be 50 times that when he was taking a normal diet with a supplement of a vitamin A concentrate.

From our standard curves, it appears that even subjects who adapt very slowly can always adapt to aperture 2 if they are given long enough. In point of fact, out of several hundred persons tested, only one, a child who did not complain of night blindness, was found who could not give readings at aperture 2, and this was probably due to the fact that the only test made could not be prolonged for more than 3 hours. Our experience of subjectively night blind cases is very limited, but the above conclusions hold good for the subclinical forms of night blindness, for the testing of which this apparatus was designed. There does, however, appear to be a threshold difference in different subjects between apertures 2 and 1, but this seems to be independent of the previous rate of adaptation. Only a small proportion of cases in which it has been tried have seen the test object at aperture 1 even after very prolonged adaptation. All but one of these showed rates of adaptation as good as or better than the general average. On the other hand, a few who gave rapid curves were tested in a darkened bedroom after dark adapting while asleep all night, and could not see the test object at aperture 1, although at aperture 2 it was immediately and easily seen.

At this point it is necessary to state that the time when the subject can say which way the E is pointing at any aperture is

not precisely the same as the light threshold represented by the illumination of the test field. There is an appreciable delay between the time when the light is first seen and the time when the E can be accurately made out. For practical purposes, however, the latter time can be considered as the time of reaching the threshold, since the interval between the curves for the absolute light threshold and that obtained using the test letter is not large, and the two curves diverge only slightly.

It has been said previously that we tried a simplified test object, the U, in an endeavour to reduce this interval. With this slide, the vision required to resolve the light seen is considerably less, and as the shape of the lighted area is less complicated, the delay between the first perception of light and the resolution of the direction of the object might be expected to be less.

The average times of 17 comparative pairs of curves with the E and with the U on 9 subjects are shown in Table III. In order to eliminate any effect of practice or fatigue, each subject (except one) was tested four times as follows: In the first session a standard curve was taken using the E test object, then the subject was "bleached" again and tested with the U. At a separate session, the same procedure was followed, reversing the order of the test objects.

TABLE III

Aperture	9	8	7	6	5	4	3	2
Average of 17 curves using "E" test object	M. S. 1 3	M. S. 2 40	M. S. 1 33	M. S. 3 4 7	M. S. 5 14	M. S. 7 20	M. S. 10 38	M. S. 13 52
Average of 17 curves using "U" test object	1 6	1 43	2 43	3 38	4 49	6 44	8 57	11 43

On comparing the curves, it will be seen that the difference between the curves is not large, except at apertures 3 and 2, where the U is resolved considerably sooner. This is to be expected owing to the relatively slower progress of the curve at its "dark" end, which causes the longer interval between the time when the test light becomes visible and that when the test object is resolved.

In future work, therefore, we intend to use the U test object. However, we see no reason to suspect any results obtained with the E, as the error involved is small and its effect is constantly in one direction.

(3) *Peripheral Vision.*—A series of experiments was made on a small number of adult subjects to determine the field through which the E could be orientated correctly. The visual fields were

first mapped out on a Skeel perimeter with the 3 mm. disc. A printed E of the same size as that used in the adaptometer was then substituted for the disc, in such a way that it could be rotated, and the fields mapped out again. A contraction of the field for all subjects of from 5° to 15° was found. In one subject only the field was contracted to between 20° and 30°, but as this subject in tests showed a rapid rate of adaptation, better than the average, this contraction of the field of vision can have had no significant effect on the test.

(4) *Effect of Visual Defects.*—The size and distance from the eye of the test letter is such that an important effect of moderate errors of refraction was not expected. The point was checked however, on 11 children used in intensive studies. Of these, 7 had normal vision, and 4 were astigmatic. Of the latter, one with moderate astigmatism showed the slowest dark adaptation curve of the series; one with high astigmatism gave a normal curve; the remaining two, one with mixed astigmatism and one with very high astigmatism, showed excellent dark adaptation curves.

In practice, it was found that many children with marked hypermetropia or myopia did better without spectacles.

(5) *The "Wave" Phenomenon.*—Kyrieleis (1938) and Müller (1931) have shown that when the course of dark adaptation is mapped out, the curve is not smooth, but in waves. We have observed that this phenomenon is often very obvious at the lower apertures, especially at the lowest, its effect being that the subject may be able to see the E once or twice, then not again until after an interval. Its importance from the point of view of technique is that one should not assume that an isolated correct response is a lucky guess. For the same reason, we do not require the three responses on aperture 2 to occur within any time limit.

An attempt was made to determine if this wave had any definite frequency by timing a series of responses on the lowest aperture, *i.e.*, when the adaptation curve was almost parallel to the base line. The time taken to change the letter is about five seconds, so theoretically a correct answer should have been obtained about every six or seven seconds. In practice there were frequently intervals varying from a few seconds to five minutes between the readings. There was no regular grouping of the responses, so that no regular rhythm of the wave could be ascertained.

(6) *Practice Factor.*—A total of 79 duplicate pairs of standard curves were taken on 14 children, both on successive days and immediate repeats. In these there was no evidence of a practice factor. The times taken to reach aperture 2 were identical in two pairs; the second was shorter in 25 pairs and longer in 52 pairs.

Repeated tests have been done on several adult and child subjects in the course of our routine work and there seems to be little or no improvement with practice, either subjective or objective. It is possible that a few subjects will not show an optimum result on a first test owing to lack of intelligence or proper co-operation. However, in our opinion, the practice factor must be negligible owing to the very close agreement obtained between curves taken on any one subject, on the same day or successive days.

(7) *Subjective Influences from the Worker.*—Many cases (mainly workers in the Rowett Institute) have been tested several times and by several combinations of observers. There is no evidence that different observers tend to obtain different results. With children as subjects, while it is not possible to improve results by suggestion beyond the optimum allowed by physiological processes, it is possible to get worse results if the child's concentration is allowed to slacken.

We believe that the method will give accurate and consistent results with any skilled observer.

(8) *Presence of Light during Course of Dark Adaptation.*—As it is impossible to have an absolutely dark room in which to conduct this test, the question arose as to whether the presence of a small amount of light during dark adaptation would affect the curve. Furthermore, Lythgoe and Phillips (1938) suggest that even subliminal light stimuli may retard the course of dark adaptation, so that the constant illumination of the test object may affect the curve.

The following experiments were therefore conducted on a small number of subjects. When the subject was fully adapted (*i.e.*, could see at aperture 2) the diaphragm was rotated back to aperture 9. The illuminated field then appears relatively brilliant. It was found that after looking at the E for 10 minutes under these conditions, the subject could still determine its position at aperture 2, immediately after the change to that aperture. Repeat tests were conducted exposing the eyes to aperture 3 in the same way, with negative results on the course of adaptation. As a further check, one subject had her standard curve taken, then was re-bleached. On going back to the adaptometer she was left looking at aperture 9 until the time had elapsed which she took to reach aperture 2 previously, when the diaphragm was rotated to aperture 2. She was able to give correct responses immediately, *i.e.*, dark adaptation had been proceeding at least as fast as previously in spite of continuous exposure to a comparatively bright light.

The evidence of Allen and Dallenbach (1938) who have studied the effect of light flashes on the course of dark adaptation is in the same direction.

Discussion

The above apparatus has, we feel, several points of advantage over any other similar apparatus yet designed, especially for routine use with children. The large test object and binocular viewing chamber make the readings reasonably easy from the subject's point of view. There is no possibility of obtaining quicker results by bringing the head nearer the test object. We also believe that it is absolutely necessary to have a test object with which the response can be checked with certainty. With our apparatus it is easy for the observer to encourage the subject to keep looking, but short of a leading question he cannot indicate the correct response. Thus with a co-operative subject the responses can represent only the course of a physiological process, except through a high degree of luck in guessing. In methods where there is no such check on veracity, and especially where a threshold response has to be determined at a specific time, it is all too easy for the observer to suggest to the subject when the response is to be expected, even by the tone of his voice.

No other published method, designed for assessing degrees of vitamin A deficiency, consistently eliminates the effect of antecedent light. The two most widely used instruments for this type of work are the biophotometer in America, and Harris's modification of the Birch-Hirschfeld photometer in this country. With these instruments the technique used limits the preliminary dark adaptation period to ten minutes, irrespective of previous light, so that on bleaching some subjects will be fully adapted, others only partially adapted. Wald, Jeghers and Arminio do not appear to allow any preliminary dark adaption before experimental bleaching. As is shown in Graph II, the dark adaptation curve, after 10 minutes' preliminary adaptation and bleaching, can vary very widely from that obtained after full preliminary adaptation, depending on previous light conditions. It must vary still more where no preliminary adaptation is allowed. It is necessary to emphasise this point, as it appears to be the major technical fault in these methods.

The bleaching apparatus used in both these techniques is also open to criticism. That used in conjunction with the Birch-Hirschfeld photometer is not of even illumination over its surface, and there is no method of controlling its intensity accurately. The bleaching screen in the biophotometer is so small that the bleaching is localised to a small area of the retina.

On both the above apparatuses the only method of checking the accuracy of a response is by recording the points first of appearance and then of disappearance of the light, with judgments

liable to subjective errors. The Birch-Hirschfeld apparatus has the additional handicap of a very small test light and no viewing chamber, so that in the dark the subject has to look for relatively tiny spots of very faint light without any material guide as to their whereabouts.

Criticism of the biophotometer has recently been forthcoming from certain sources (Palmer and Blumberg, 1937; Palmer, 1938; Gridgeman and Wilkinson, 1938; Isaacs, *et al.* 1938). We believe that the above points of technique may be partly responsible for the dissatisfaction expressed by these workers.

It should be noted that the method here described differs from most others in using white light for the test. This will be discussed in the next paper of this series in which the results will be reported of an investigation into rates of dark adaptation obtained in groups of subjects whose diets differed widely.

We take this opportunity to express our indebtedness to Professor J. A. Carroll for granting us facilities in the Department of Natural Philosophy of the University of Aberdeen for development and standardisation of the adaptometer.

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A NOTE ON THREE CASES SHOWING "CROCODILE TEARS" AFTER FACIAL PARALYSIS

BY

L. H. SAVIN

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IN 1931 a bricklayer's labourer attended the Royal Eye Hospital complaining that if he ate appetising food, one eye always watered. Two years previously he had had severe facial paralysis on that side, with involvement of taste along the same side of his tongue. Recovery had been slow in spite of much electrical treatment. On examination the gross movements of the face were found to have recovered fairly completely, but when he whistled or smiled there was considerable twitching and fibrillation elsewhere in the face. On his first visit to the hospital he had just lunched, and the eye did not water when he ate a biscuit. The lower punctum was in good apposition with the globe, and the nasal duct was patent. He was instructed to omit his lunch before the next visit, when eating a biscuit produced copious watering of the eye. It was thought that the phenomenon had some connection with salivation, but the condition remained unexplained and unrelieved.

Search of the literature shewed several reports of the phenomenon, which has been aptly named "crocodile tears" ("Krokodils-tränen," Bogorad, 1928), after the hypocritical tears legendarily ascribed to these reptiles before devouring their victims (see Sir John Maundeville's Travels, c. 1400). Cases have been described by Bogorad (1928), Kaminsky (1926), and Tumarkin (1936). Tumarkin, who reported 13 cases, seems to have given the most reasonable explanation. Normally in Bell's palsy the site of the lesion in the facial nerve is somewhere near the stylomastoid foramen. In more severe cases the nerve may be affected inside the temporal bone in the aqueductus Fallopii. Certain clinical signs may indicate such involvement. If the great superficial petrosal branch from the geniculate ganglion is affected, there may be absence of lacrymation at the first onset of the paralysis instead of the usual mechanical epiphora from orbicularis paralysis. Some at any rate of the secretory fibres of the lacrymal gland pass from the geniculate ganglion of the facial along the great superficial petrosal nerve, and after this has been joined by the great deep petrosal nerve, the secretory fibres pass along the Vidian nerve to Meckel's ganglion. Thence they may take two alterative routes, either passing along the two sphenopalatine nerves, the superior maxillary nerve, the temporomalar (zygomatic) nerve, its communicating branch to the

lacrymal gland, and so to the lacrymal gland; alternatively orbital branches may connect Meckel's ganglion directly to the lacrymal gland.

Clinical signs are less obvious when the secretory fibres to the parotid are involved. These fibres pass through a communicating branch from the geniculate ganglion to the lesser superficial

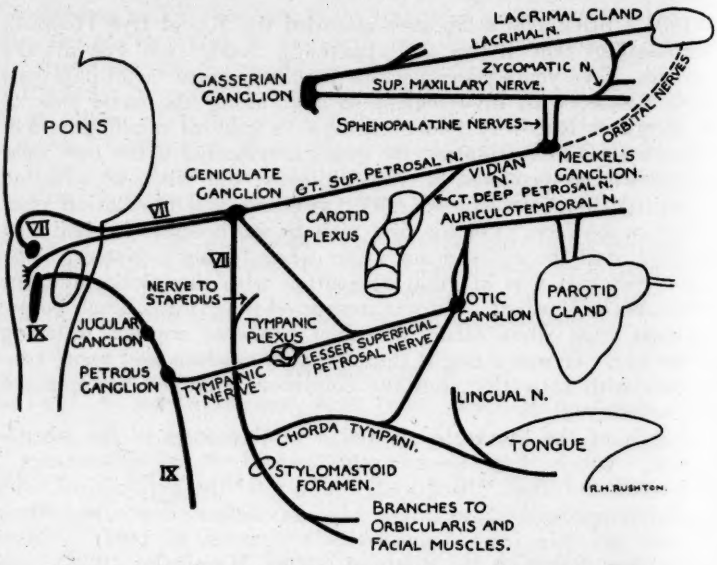

Diagram of the nerves involved in the production of "crocodile tears."

petrosal nerve and the otic ganglion (or alternatively by a branch from the chorda tympani to the otic ganglion). Thence two twigs pass to the auriculo-temporal nerve, through the glandular branches of which the secretory fibres reach the parotid gland.

If the nerve to the stapedius is involved, the patient may complain of hyperacusis, in which certain sounds acquire an unpleasant jarring timbre.

If the chorda tympani is affected, taste will be lost in the anterior two-thirds of the same side of the tongue.

Later when the facial nerve regenerates, Tumarkin states that the newly-growing axis cylinders do not always extend along their original neurilemmal pathways. Hence the phenomenon of synkinesia, where for instance the patient may unconsciously contract the muscles of the nose and mouth, when shutting the eye. Where regenerating parotid secretory fibres accidentally

pass along the paths to the lacrymal gland, a form of synkinesia will occur in which the patient has to mop his eye when salivation occurs during meals. These " crocodile tears " may produce a troublesome secondary blepharitis. Such lacrymation is distinguishable from the mechanical watering caused by an everted punctum from orbicularis paralysis.

Since reading Tumarkin's papers I have watched for the phenomenon and now report two additional cases.

A barrister's wife developed right facial paralysis in 1925 when she was aged 29 years. She does not remember if taste was affected in her tongue, though she can describe vividly her initial difficulties in mastication, eating and drinking. Synkinesia is now most obvious in her lower lid which fibrillates when her face moves. Her eye streams with water when she eats; so much so, that she has for years disliked to dine in a restaurant away from home, as she is usually annoyed by some officious waiter who solicitously asks whether she has something in her eye.

A printer seen recently with " crocodile tears " had right facial paralysis first in 1908, and attended hospital for several years for electrical treatment. His chorda tympani nerve was evidently affected, as for some years the side of his tongue " felt as if made of indiarubber." His right eye waters so severely on eating, that for years the patient has retired to a secluded spot for his mid-day meal, so as to avoid the chaffing of his fellow workmen. His face is drawn up and contracted on the affected side. Synkinesia is very obvious as fine fibrillary twitching over most of the right side of the face, when he shuts his right eye, whistles, or smiles.

The best method of treatment deserves consideration. The two patients just described were advised to undergo excision of the palpebral portion of the lacrymal gland on the affected side. Both refused on the reasonable grounds that they had become habituated to, and tolerant of their peculiarity. They maintained this attitude when offered an alternative suggested by my oto-rhinological colleagues, Mr. Negus and Mr. Cawthorne. Since the parasympathetic secretory supply to the lacrymal gland passes through Meckel's ganglion, it should prove possible to block temporarily the secretory fibres by cocainization between the posterior end of the middle concha and the lateral wall of the nose, employing a cotton wool-covered probe dipped in cocaine paste or solution. If this simple measure temporarily abolished " crocodile tears " when eating, permanent relief might be expected from blocking Meckel's ganglion with alcohol after the Sluder technique: this would be preferable to the somewhat mutilating procedure of excision of the palpebral portion of the lacrymal gland. Ruskin (1928) has reported lessening of lacrymation after injection of the sphenopalatine ganglion in a patient with obstructed nasal duct.

A similar patient, in whom the ganglion was recently blocked by Mr. Negus with cocaine, could notice no benefit even of temporary nature. We did not think the experiment conclusive, as there was in this case concurrent conjunctivitis.

I wish to thank Messrs. T. Cawthorne, A. D. Griffith, V. E. Negus, S. Nevin, A. Sorsby, and I. A. Tumarkin for kind advice and explanations.

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ON THE LOCALISATION OF THE MERIDIAN DETERMINED BY PERIMETRIC MEASUREMENT ON THE OUTSIDE OF THE SCLERA

BY

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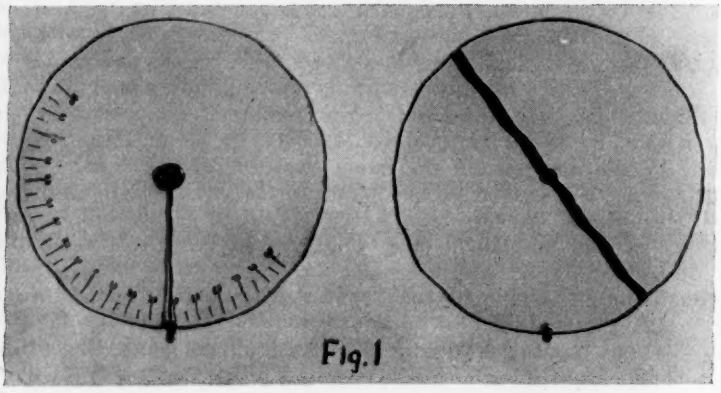
THE transfer of the meridian determined by the angle which the plane of the arc of the perimeter forms with the horizontal is not simple. Being done at sight gives only approximate results. Then several authors, Lindner, Zykulenko and others, use for this purpose complicated instruments similar to a goniometer, which



are transferred to the patient's eye. A much more simple method is that of Klein, who uses as his fixation light on the perimeter a filament lamp which rotates with the arc, the image of which is then projected upon the eye, see Duke-Elder's Text-Book of Ophthalmology, Vol. II.

I take the liberty to offer a new simple method, founded on keratoscopic examination, Fig. 1. Instead of concentric rings across the centre of the disc is drawn a black line, which passes through the zero, marked on the back of the disc. Here is placed, similar to the keratoscope of Weve, a metallic rod with weight—a plummet—fastened to the movable frame of the magnifying-glass, 6·0 D., placed in the hole of the disc. The rod determines a perpendicular, 0°, or a declination.

The proceeding is simple: we set the metallic rod suitable to the site of the arc of the perimeter, in the Fig. 52° from the horizontal, and holding the disc parallel to the iris or suspending it on the free end of the rod at a distance of about 12 cm., the diameter of the disc is 33 cm., we look at the corneal image. The image of the centre of the disc shall be brought to the centre of the cornea. The image of the black line indicates the meridian of longitude, determined by perimetric measurement, in which the retinal landmark is situated. The two points on opposite sides of the limbus are marked with some indicator.

ANNOTATION

Recent changes in some Continental Ophthalmological Journals

The *Zeitschrift für Augenheilkunde* now appears as "Ophthalmologica." It is published as an international journal of ophthalmology at Basel and contains articles in English, French and German. The list of editors is world-wide and we note that for Britain these are A. J. Ballantyne, Sir S. Duke-Elder, Mr. C. B. Goulden and Miss Ida Mann. We understand that the *Archiv. für Ophthalmologie* and the *Archiv. für Augenheilkunde* have been recently amalgamated. We do not know what facts underlie these changes. The causes of amalgamation of the two *Archiv.* may possibly be financial. Like Mr. Squeers, when Bolder's father was two pound ten short, they may have had disappointments to contend against. The financing of an ophthalmological journal is never an easy matter in any country. Union usually means strength and under conditions as they are to-day in Central Europe there may not be sufficient material to enable two large journals such as the

two *Archiv.* to keep going. If so an amalgamation is the obvious way in which to meet the difficulty. But for the *Zeitschrift* to change its name and country of publication appears to us to suggest that the editorial department feel that they need a freer hand in matters for publication relating to their own subject. It will be a poor day for science of every kind if methods of control are allowed to interfere with the free interchange of knowledge; for science is truly international and independent of race and creed.

ABSTRACTS

I.—MEDICAL OPHTHALMOLOGY

(1) **Naffziger, Howard C. (San Francisco).—Progressive exophthalmos associated with disorders of the thyroid gland.** *Annals of Surgery*, October, 1938.

(1) **Naffziger** states that before 1931 a review of the literature showed no concurrence of opinion as to the reasons for the progressive exophthalmos occasionally seen following thyroidectomy. He then had a case in which operative treatment was successful and he was able at the same time to investigate the pathological changes involved. Since then a number of other cases have been investigated and the pathological changes have been verified by numerous observers.

"Granting that our use of the term exophthalmos is loose and inaccurate, one cannot say much more than that, after an operation for goitre, the apparent exophthalmos disappears in about one-half of the cases and lessens in an additional 15 or 20 per cent. In others it may remain unchanged, but in a few there will be progression of varying grades. This progression may continue slowly, causing no more difficulty than that involved in an unsightly appearance of the patient; progression may halt at any time and it is, fortunately, in only a very small number that it will progress to a danger point."

The cases in which operation has been performed by other surgeons together with eight of the author's make a total of 31. In this series two deaths occurred and four results were considered poor. In the author's series of eight cases the age varied from 28 to 53 years. Four were females, four males. All had marked thyrotoxicosis at some time.

Examination of pieces of tissue taken from the orbits at the time of operation proves that the ocular muscles show varying degrees of degeneration, fibrosis and cellular infiltration.

In some cases additional biopsies of muscle were taken from other parts of the body but no such changes were found in them.

Naffziger gives the case report of a man aged 39 in 1930 who complained of diplopia. Six months later exophthalmos of the left eye was noted. Enucleation became necessary. Two months later the remaining eye began to show exophthalmos, and evidence of thyrotoxicosis appeared. The basal metabolic rate was plus 24, and later in the same month thyroidectomy was performed. In January, 1932, the basal metabolic rate was plus 6. Yet the exophthalmos progressed and by September of that year vision was blurred and the optic disc was swollen 4 D., retinal haemorrhages being also present.

When admitted to hospital the basal metabolic rate was minus 13. There was great retrobulbar resistance. Exudates had appeared in the fundus and the exophthalmometer reading was 31; visual acuity, 120/200. Eye movements were limited and the field showed contraction. Operation was performed in October, 1932. There was a reduction of 4 mm. in the exophthalmometer reading while the man was still in hospital. The fundus condition rapidly cleared up and vision improved. Six years later, the patient considered the eye to be completely restored in vision and movement, although it is still more prominent than normal.

Details of the author's operation for this condition will be found in the *Journal of the American Medical Association* of August, 1932. Here he says: "We now remove not only the area of bone over the roof of the orbit, but carry the removal of bone lateralward into the temporal fossa and up close to the orbital rim, removing that portion of the lateral wall down to the antrum. Posterior to this, the removal of bone is continued back into the middle fossa, removing the postero-lateral portion of the orbit down to, and about, the orbital fissure. When choked discs are present, the optic foramina are unroofed."

Since he has used this extended removal of bone he has found that the immediate recession of the eyes has been much greater and post-operative hazard lessened.

A good drawing of a skull illustrates the area of bone to be removed on one side and the orbital area on the other side after the removal of bone. There are micro-photographs of the pathological changes present in the eye muscles with photos of the patient whose case is recorded at length and photos of three other patients before and after operation to show the very striking improvement he has obtained.

A bibliography of nine items concludes the paper and a short description of the discussion that followed its delivery.

R. R. J.

(2) **Whiteside, W. Carleton (Edmonton).—Leber's hereditary optic neuritis through six generations—a sterilization problem.** *Canadian Med. Assoc. Jl.*, October, 1938.

(2) **Whiteside's** case came to him for the purpose of being sterilized; she knew of her family history and had been told that if she bore male children they would become blind between the age of thirteen and thirty.

Her family tree contained twenty-three males who had become blind. Whiteside demanded all details of intermarriages and family names; and after thorough "investigation of the afflicted cases by consultation and correspondence, the accompanying genealogical table was produced, the summation of which would indicate that sterilization of this female patient (represented by the rosette circle in table) was contra-indicated, and that the patient could propagate *ad libitum* without the fear of her offspring becoming blind."

In searching the literature he was unable to find an extensive review of a long genealogical table such as is represented here. The condition is a problem of preventive medicine, sterilization being the only method of eradicating the condition.

In his summary the author pleads for "continued co-operation between the ophthalmologist and the surgeon as a means of eradicating the social and economic problem of this condition."

R. R. J.

(3) **Carroll, F. D. and Goodhart, R. (New York).—Acute alcoholic amaurosis.** *Arch. of Ophthal.*, November, 1938.

(3) **Carroll** and **Goodhart** during one year have observed no fewer than six cases of total but temporary blindness associated with acute poisoning due to ethyl alcohol. In their paper they give a detailed account of four of these. The condition develops during a period of acute intoxication, but a necessary pre-requisite seems to be addiction to alcohol. It may also be significant that in all the six cases there was a previous history of trauma to the head.

One feature of the condition is that although the patients are totally blind, normal pupillary reactions are retained. Recovery is fairly rapid—in one case 12 hours—but may be apparently delayed by the development of hysterical blindness. The site or character of this lesion has not been determined but it is probably central. Although rare (0·05 per cent. of all instances of ethyl alcohol poisoning) the condition is important, because of the excellent prognosis which can be given, as contrasted with the poor prognosis in methyl alcohol poisoning.

F. A. W-N.

(4) **Hanum, S. (Copenhagen).—Diabetic retinitis: Clinical studies of 195 cases of retinal changes in diabetics.** *Acta Ophthal. Suppl.*, Vol. XVI, 1939.

(4) In a study of 966 diabetic patients, 195 were found by **Hanum** to suffer from retinitis. The highest incidence occurred in the fifth and sixth decade of life, and there was a definite relationship between the duration of the diabetes and the occurrence of retinal changes. In this series women were more prone to the affection than men, the ratio being 5:2. There was no relationship between the severity of the diabetes and the incidence of retinitis, though there seemed to be evidence that more diabetics who had received little or no insulin treatment were affected than those who were treated with insulin. Paradoxically enough it would also appear that diabetic retinitis is more common now-a-days than it was before the introduction of insulin. In discussing fundus appearances the author stresses what he calls the proliferative type, characterized by massive exudation into the vitreous and even retinal detachment. He holds that in such cases there is a loss of capillary resistance and a diminution of the ascorbic acid content of the blood. Some success by treatment with ascorbic acid, lemon juice and citrin (vitamin P) is recorded. The author could find no definite evidence of renal impairment as a factor in diabetic retinitis, nor could he substantiate the view that hypotension is an essential factor. In 28 cases of retinitis the retinal blood pressure was determined by ophthalmo-dynometry and in some 75 per cent. it was found to be raised—a finding of no clear significance. On a statistical evaluation the author concludes that diabetics with retinitis have a lower life expectation than patients not showing this complication.

ARNOLD SORSBY.

(5) **Feigenbaum, A. and Rachmilewitz, M. (Jerusalem).—The significance ot retinal findings in hypertensive vascular and renal disease.** *Acta Ophthal. Orientalia*, Vol. I, p. 65, 1938.

(5) **Feigenbaum** and **Rachmilewitz** correlate fundus studies with medical findings in 168 patients of whom 93 were of essential (benign) hypertension type, 17 with malignant hypertension and 58 with nephritis. The fundus of the essential hypertension group showed characteristic changes of "fundus hypertonicus" in 89 per cent. consisting mainly in general reduction in calibre with arteriovenous compression; tortuosity and irregularity in calibre were present in a smaller proportion.

The 17 patients with malignant hypertension (with or without renal manifestations) presented the same alterations in the arterioles, only far more frequently and much more pronounced than in the benign hypertension group. An important and characteristic finding

in the malignant hypertension group was engorgement of the veins, retinal exudates, haemorrhages and oedema including papilloedema in many instances.

In patients with chronic nephritis the predominant feature was general arterial constriction while the other arteriolo-sclerotic changes characteristic of non-nephritic hypertension as well as venous hyperaemia were insignificant. This essential difference between the nephritic and the non-nephritic type may serve as a basis for differentiation between the two groups even in the advanced stage of the so-called "retinitis albuminurica," and may at the same time indicate a different pathogenesis. The correct term for "albuminuric retinitis" of the malignant type should be "arteriolo-sclerotic retinopathy."

ARNOLD SORSBY.

(6) **Bangerter, A. (Bern).—An unusual case of diabetic cataract. (Ein ungewöhnlicher Fall von Cataracta diabetica).** *Ophthalmologica*, Vol. XCVI, p. 98, 1938.

(6) **Bangerter** reports on the case of a girl, aged 11 years, suffering from diabetes and showing cataract in both eyes. The cataract was punctate in appearance, but scattered throughout the lens, most marked in the sub-capsular area. There was also a flat cloud-like opacity at the posterior pole containing glistening dots. Attention is drawn to a similar case published by Nobécourt, and it is argued that in addition to the classical sub-capsular type there is this other form of diabetic cataract affecting the lens diffusely.

ARNOLD SORSBY.

(7) **Jancke, G. (Köln).—The engorged retina as an expression of right-sided heart dysfunction. (Die Stauungsnetzhaut als Ausdruck der Beteiligung der Netzhautgefässe bei Versagen des rechten Herzens).** *Klin. Monatsbl. f. Augenheilk.*, Vol. XCIX, p. 756, 1937.

(7) **Jancke** describes several cases of congested retina resembling "cyanosis retinae," but associated with emphysema and enlargement of the right side of the heart, rather than with polycythaemia. He notes the interesting fact that papilloedema may occur secondarily to venous congestion.

D. R. CAMPBELL.

(8) **Orzalesi (Florence).—Arterial retinal pressure in toxic amblyopia. (La pressione arteriosa retinica nell'ambliopia alcoolico-nicotinico).** *Boll. d'Ocul.*, November, 1938.

(8) In recent years research has been made into the condition of the circulation in the optic nerve in the various forms of optic

atrophy. It is known that in some cases vision is preserved in glaucoma even when the intra-ocular pressure is high. **Orzalesi** suggests that this may be correlated with high tension in the retinal arteries; on the other hand that tabetic atrophy may to some extent depend on low retinal arterial pressure. The way in which intoxication leads to amblyopia is not known; it has been suggested that the toxic material acts on the ganglion cells and their axis cylinders directly; others have assumed some alteration of the blood supply; the author has examined the arterial pressure in a number of these cases and finds that in the majority there is low arterial pressure, but holds that this cannot be the sole cause of the atrophy though it may be an adjuvant.

HAROLD GRIMSDALE.

(9) **Pringle, J. Hogarth (Glasgow).—Traumatic meningeal haemorrhage with a review of seventy-one cases.** *Edin. Med. Jl.*, November, 1938.

(9) **Pringle,** in discussing the diagnosis as to whether the case is one of extra or subdural haemorrhage, states that personally he has not derived much assistance from the state of the pupils, and that their significance as a test loses value from its uncertainty. Nor did he find that an examination of the fundi in these cases assisted in any special degree.

The author, however, is emphatic in his praise of the value of the "Macewen pupil" in differentiating cases of subdural haemorrhage from the state of alcoholic coma. He states that in his experience this test has always been positive in alcoholic coma, and forms in his opinion, a most valuable diagnostic sign.

In discussing the subject of subdural haemorrhage and its relation to haemorrhage into the sheath of the optic nerve, the author describes how in five fatal cases blood was found, at post-mortem examination, in the sheath of the intra-orbital portion of the nerve, but not on the cut-surface of the nerve as made in the removal of the brain; this goes to prove that the blood in the sheath had arisen from the vessels of the nerve or its sheath, and was not blood forced into the sheath from the subdural space.

G. I. SCOTT.

(10) **Agnello (Addis Ababa).—Angioneurotic oedema and allergic conjunctivitis. (Edema angioneorotico e congiuntivite allergica).** *Boll. d'Ocul.*, October, 1938.

(10) Of all ocular structures the conjunctiva is that chiefly affected by allergic manifestations. Conjunctivitis is a very frequent concomitant of asthma; "spring catarrh" is generally admitted to be an allergic state. **Agnello's** patient had suffered from childhood

from urticaria of an allergic nature. Later in life she became subject to paroxysmal tachycardia and to facial oedema, which almost closed the palpebral fissure. The exact cause of these symptoms, which were certainly allergic, was not discovered.

HAROLD GRIMSDALE.

(11) **Streiff (Geneva).—Angioid streaks in the retina. (Le strie pigmentate moniliformi della corio-retina).** *Boll. d'Ocul.*, October, 1938.

(11) **Streiff** gives the history of three cases which showed this condition. All three were past middle life and two were slightly myopic. All showed arterio-sclerosis. The streaks begin usually at a little distance from the disc; they may be linear or made up of dots in a line covering the subjacent choroidal vessel, which is frequently sclerosed. There are often associated with them, haemorrhages in the retina; one of the author's cases showed a very large haemorrhage in the macular region of one eye, which however, did not show any angioid streaks. The author holds that these streaks, made up of dots, and resembling strings of beads must be separated from the angioid streaks of Groenblad and Strandberg.

HAROLD GRIMSDALE.

II.—CORNEA

(1) **Kentgens, S. K. (Utrecht).—Vitamin A therapy in corneal lesions. (Ueber Vitamin-A-Therapie bei Hornhauterkrankungen).** *Ophthalmologica*, Vol. XCVI, p. 3, 1938.

(1) On an analysis of the vitamin A content of the blood in a series of patients with corneal affections and on the results obtained with vitamin A therapy by mouth and locally, **Kentgens** concludes that this method of treatment is particularly valuable in cases of herpes of the cornea, the duration of the affection being reduced by half (from 584 days to 249). Superficial punctate keratitis, marginal ulcers and catarrhal ulcers are also favourably affected. The results obtained in two cases of Mooren's ulcer and in ophthalmia neonatorum with corneal involvement were also satisfactory.

ARNOLD SORSBY.

(2) **Rintelen, F. (Basle).—Fixing the graft in keratoplasty. (Ueber Lappensicherung bei der durchgreifenden optischen Keratoplastik).** *Ophthalmologica*, Vol. XCVI, p. 155, 1938.

(2) **Rintelen** describes a method of fixing the corneal graft by covering it with gutta percha membrane and securing this cover

by means of cross threads from limbus to limbus. Good fixation is obtained and the graft can be viewed through a small opening in protective gutta percha membrane.

ARNOLD SORSBY.

(3) **Friede, R.—A clinic of active optical keratoplasty. (Zur Klinik der durchgreifenden optischen Keratoplastik).** *Klin. Monatsbl. f. Augenheilk.*, Vol. CI, p. 1, 1938.

(3) **Friede** gives a careful review of 78 cases of keratoplasty. The original article should be consulted by those interested in the operation. The author obtained the best results in cases of scrofulosis, herpes and ulcer of the cornea and least success after ophthalmia neonatorum. The presence of anterior synechiae or a total leucoma lessens the chance of obtaining a clear graft. In the last condition pre-operative glaucoma is common and may require treatment, *e.g.*, by an extensive cyclodialysis. The most favourable size of graft is 4-5 mm. The cornea of a sarcoma eye is still the one most used while those taken from the cadaver give good results. The author advises the use of atropine in making a small graft and eserine when the graft is larger than 6 mm. Removal of a cataractous lens should be done after the corneal transplantation.

D. R. CAMPBELL.

III.—MISCELLANEOUS

(1) **Nicholls, John V. V. (Montreal).—A case of granuloma of the lacrymal canaliculus.** *Canadian Med. Assoc. Jl.*, December, 1938.

(1) **Nicholls**, while working with Sir Henry Holland at Karachi, observed an unusual case. The patient was a female aged about 60 years with a small mass, the size of a pea, in the margin of the right upper lid. The lump was situated about 2 mm. to the nasal side of the punctum and had been present for three years. It was found to be attached to the skin over it and to the deeper structures. There were scars in the conjunctiva of the upper lid, apparently resulting from a mild attack of trachoma. The mass was easily removed under local anaesthesia. It was non-encapsulated. The tissue removed was examined in the laboratory of Moorfields Eye Hospital. Sections showed a non-encapsulated mass of granulation tissue surrounding and compressing the canaliculus. It was mainly composed of a stroma of fine fibrous tissue with lymphocytes and plasmocytes. A few endothelial cells were present with the small giant-cell containing not more than four large oval vascular nuclei.

The mass was practically avascular, contained no organisms or foreign body and no necrotic areas were present.

In the author's opinion this was probably a trachomatous affection of the canaliculus.

A high power section of the mass accompanies the paper.

R. R. J.

(2) **Dubois-Poulsen.—The spheno-palatine ganglion and the eye. (Le ganglion spheno-palatin et l'oeil).** *Ann. d'Ocul.*, Vol. CLXXIV, p. 217, April, 1937.

(2) **Dubois-Poulsen** is known as a protagonist of nasal cocainisation, a method of treatment which at present is much discussed in French ophthalmological papers. A swab of cotton wool soaked in 10 per cent. solution of cocaine and left for an hour in contact with the posterior part of the middle turbinate is credited with curing a large number of painful eye conditions.

The immediate object of the cocaine application is to bring on the anaesthesia of the spheno-palatine ganglion.

The author gives a detailed description of the anatomy of the ganglion and of its afferent and efferent fibres. It is a sympathetic ganglion and controls the lacrymal gland and the mucous glands of the nasal cavity. A stimulus acting on the conjunctiva or on the nasal mucous membrane sets in operation a reflex arc which passes up the trigeminal fibres and is probably relayed to the multipolar cells of the nucleus of the facialis. The centrifugal path begins in the sympathetic nervus intermedius of Wrisberg and follows the course of the facial nerve to the geniculate ganglion. From this ganglion the impulse travels in the greater superficial petrosal nerve. This nerve joins the sympathetic deep petrosal nerve and runs through the Vidian canal to the spheno-palatine ganglion. Efferent fibres of this ganglion convey the impulse to the lacrymal gland. At the same time, the stimulation of the ganglion induces a dilatation of the conjunctival, nasal and facial vessels. The nose as well as the eye begins to water profusely. Sometimes the patient complains of paraesthetic sensations in distant areas, *e.g.*, in the back of the neck and in his legs. The diastolic pressure in the central artery is increased without a change in the general circulation becoming noticeable. The tension of the normal eye remains unaltered. The central visual acuity is not reduced, however the visual field may become restricted concentrically. The blind spot invariably becomes enlarged and the scotoma which corresponds to the course of the larger central vessels can easily be mapped out. In addition, the stimulation of the ganglion has a very marked psychic effect, which varies according to the personality of the patient. It may bring on euphoria in balanced people and hysterical fits in emotional women.

The anaesthesia of a ganglion with such activities can be expected to be efficient in the treatment of many ocular troubles.

In profuse watering of the eye not due to lacrymal stenosis the cocainisation of the ganglion brought on a cure in eighteen cases out of twenty-three.

The same treatment was very successful in cases of sub-acute conjunctivitis which could not be ascribed to a bacterial infection. These affections, which are often unilateral, cleared up in four or five days after three or four months duration.

The author records rapid improvement of superficial episcleritis, but the nodules of deep scleritis proves refractory to the treatment.

Superficial punctate keratitis with subepithelial infiltrations near the limbus healed rapidly after the nasal treatment. A similar success could be stated in a type of kerato-conjunctivitis resembling trachoma.

Even patients who could not be cured by cocainisation obtained relief from pain, photophobia, watering and blepharospasm. Patients who have a poor corneal sensation seem to respond more readily to the ganglion anaesthesia. Apparently, the corneal lesion is not entirely due to the lack of sensation and consequeut exposure to injuries. The trophic rôle of the sympathetic fibres cannot be denied. Indeed, no paralytic keratitis results if the fifth nerve is cut above or at the level of the Gasserian ganglion before the sympathetic fibres have joined it.

Another affection which clears up after ganglion anaesthesia has been labelled the syndrome of the naso-ciliary nerve. This name has been given by Charlin to a triad of symptoms: inflammation of the anterior segment of the eye, neuralgic pain at the point of exit of the external branch of the naso-ciliary nerve and profuse nasal watering. Charlin suspected a neuritis of the naso-ciliary nerve. He treated this syndrome successfully by cocainisation of the lower turbinate and the lower meatus. However, it is more probable that this treatment is efficient by abolishing axon-reflexes and prevents the excitation of the nasal branch from being conveyed antidromically to the ocular branch of the nerve. Charlin's syndrome can be cured more efficiently by blocking the nervous path at the ciliary ganglion. For this purpose the author recommends the injection of 2 c.c. novocaine followed by 2 c.c. of 40 per cent. alcohol.

The results of nasal cocainisation in herpes zoster are not so brilliant. The excruciating pain of a patient could be relieved by an injection into the spheno-palatine ganglion. The nasal painting had an insignificant result.

The ganglion anaesthesia has given good results in various types of headaches. It is an excellent treatment for Sluder's syndrome which is described as a triad of orbital and periorbital pain, so-called accommodative asthenopia and a sensation of the eyeball being too

big for the orbit. There are frequent exacerbations of the pain. The condition is probably due to hyperactivity of the spheno-palatine ganglion. The author records two cases of migraine in which the nasal treatment had been successful. Most patients suffering from migraine were not much improved.

In glaucoma the cocainisation was followed by a bilateral decrease of the hypertension. The decrease was more marked on the side of the application. A much more powerful method to reduce ocular hypertension is the injection of 40 per cent. alcohol into the ciliary ganglion. In this way each case of acute glaucoma can be brought down to a subacute stage before operating on it. Again, the injection into the ciliary ganglion is more efficient than nasal painting in relieving the glaucomatous pain.

In optic neuritis the opening of a nasal sinus has often proved successful even in cases in which the sinus was found to be healthy. As the sphenoid sinus is situated in the immediate neighbourhood of the spheno-palatine ganglion, the success of the operation is probably due to accidental interference with the fibres which run from the ganglion to the optic nerve and which are supposed to control the retinal circulation.

The anaesthesia of the ganglion has been very useful not only in well defined conditions. It is the method of choice in treating neurotic people who like to choose the eye as focus of their troubles.

HUMPHREY NEAME.

(3) **Minton, J. (London).—Industrial eye injuries.** *The Practitioner*, December, 1938.

(3) **Minton** gives an account of industrial eye injuries which should be of use to general practitioners. He states that these are on the increase. They are responsible for 10 per cent. of the blind in this country. Their high incidence is due to:—(1) The non-provision of safety measure by many employers; (2) complete negligence of the workmen in using the safety measures provided.

The problem should be tackled from two sides:—(1) The employers should be made conscious of the necessity of providing safety measures. The new Factories Act will enlarge the scope of the compulsory use of protective measures. (2) A determined fight should be made to educate the workmen and to encourage them to overcome all their prejudices against wearing goggles and other protective devices.

When work is being done away from the factory, without the protective appliances at hand, it ought to be possible for intelligent workmen to supply themselves with them at a cost within their competence. On more than one occasion the reviewer has offered

a protective veil, useful against light flying particles, to a workman who has accepted it with alacrity. Such a veil costs only twopence.

A. F. MACCALLAN.

(4) **Jaeger, E. (Traunstein).—Investigation of school-myopia. (Untersuchungen ueber Schulmyopie).** *Klin. Monatsbl. f. Augenheilk.*, Vol. CI, p. 205, 1938.

(4) **Jaeger** gives the result of an annual investigation of 350 girls aged 10-16 years, and 150 boys, aged 10-18 years, taken over a period of ten years. He finds that myopia most commonly occurs at 12 years old and rarely shows an increase after the age of 15. Increase in the number of dioptres of myopia occurs rapidly in the second and third years of school life. Myopia appears to be correlated with age and physical development rather than with the mental requirements of school. Environment probably exercises an additional influence.

D. R. CAMPBELL.

(5) **Motolese (Ancona).—Papilloedema and bilateral paralysis of the sixth nerve after spinal anaesthesia from novocaine). (Paralisi del retto esterno bilaterale associata a papilla da stasi da rachianestesia novocainica).** *Boll. d'Ocul.*, August, 1938.

(5) Disturbance of the ocular muscles is not an uncommon accident after spinal anaesthesia. Changes in the fundus are much less frequent. **Motolese** reports a case in which bilateral paralysis of the external rectus was complicated by loss of visual acuity due to atrophy following papilloedema. Unfortunately he did not see the patient for nearly two months after the operation, by which time much damage had been done to the nerve. He holds that if the pressure had been relieved by lumbar puncture earlier, there would have been a good chance of recovery.

HAROLD GRIMSDALE.

(6) **Santoni (Naples).—A glioma of the optic nerve. (Glioma primitivo del nervo ottico).** *Boll. d'Ocul.*, November, 1938.

(6) The patient was a child, aged 10 years, and the tumour had been noted for some eight years, as the eye was exophthalmic at the age of two years. At the age of four years, the child fell down stairs and the exophthalmos was reduced but the sight was lost in that eye. There seems little doubt that at that time the eye was ruptured and emptied. Since the exophthalmos reappeared, the parents consented to operation (which had been advised before but refused). There was removed a mass in a capsule filling the greater part of the orbit. The tumour was largely undergoing mucoid degeneration. It was

possible to find in some places oligodendrocytes. **Santoni** considers the tumour as probably belonging to the group of oligodendrocytic gliomata.

HAROLD GRIMSDALE.

(7) **Georgariou, Peter M. (Halle Salle).—Cholesterinosis bulbi.** *Arch. f. Ophthal.*, Vol. CXXXIX, p. 32.

(7) Cholesterin crystals occur in the eye under two different forms, for which, as explained here by **Georgariou,** there are two different methods of production:—

(*a*) Partial lipoidosis oculi, which has been described under various names, is characterised by a local infiltrative fatty degeneration in the iris epithelium and the endothelium of the posterior surface of the cornea and secondarily in the underlying basal tissue. The appearance of individual cholesterin crystals is a later secondary development.

(*b*) In cholesterinosis bulbi, a case of which is here described with the cholesterin in the form of typical rhomboid crystals filling the anterior chamber, a change in the composition of the intraocular fluid takes place, following repeated inflammation in the ciliary body, with a greater permeability of the vessels and the escape of more protein and colloids which undergo a process of "chemical digestion" in the anterior chamber. The tissue fluid between the coats of the eye also suffers a disturbance in the colloids present in it, which leads to the exudation of numerous cholesterin crystals in the tissue spaces.

Why this deposit of crystals does not occur in all cases of cyclitis is a question that is not answered, but it is hinted that the traumatic form of cyclitis may have a specific influence on their formation.

The author appends a full bibliography on the subject.

THOS. SNOWBALL.

(8) **Németh, L. (Budapest).—The constitution of the glaucoma patient. (Ueber die Konstitution der Glaukomkranken).** *Klin. Monatsbl. f. Augenheilk.*, Vol. CI, p. 222, 1938.

(8) **Németh** investigated 100 cases of glaucoma in Berlin and Budapest. He was unable to find any particular type of physique associated with primary glaucoma, but glaucoma simplex appeared to be common in asthenic individuals, whereas acute inflammatory glaucoma occurred in broad, short ("pyknic") individuals. Asthenics tend to have a lower blood pressure and are subject to earlier and more rapidly progressive glaucoma. They require rest of the nervous system, whereas "pyknics" are subject to vascular crisis.

D. R. CAMPBELL.

(9) **Zondek, H. (Jerusalem).—On the question of retrogression of bitemporal hemianopsia under internal therapy.** *Acta Ophthal. Orientalia*, Vol. I, p. 88, 1938.

(9) **Zondek** recalls that in some cases of salt-water obesity the increase of intracranial pressure causes bitemporal hemianopsia, probably due to swelling of the tissues in the vicinity of the chiasma. A regimen directed towards the dehydration relieves the symptoms.

ARNOLD SORSBY.

(10) **Ascher, K. (Prague).—Regressive changes induced by X-ray in an epithelial rest in the anterior chamber. (Epitheleinsprossung in die Vorderkammer. Regressive Veränderungen und Heilung nach Röntgenbestrahlungen).** *Ophthalmologica*, Vol. XCVI, p. 29, 1938.

(10) **Ascher** describes a characteristic case of an epithelial cyst in the anterior chamber which responded quickly and decisively to X-ray treatment: three doses of 400 r and two doses of 300 r, a total of 1800 r being given within 24 days.

ARNOLD SORSBY.

(11) **Simonelli (Pisa).—The function of Vitamin A in vision. (La funzione della vitamina A nel meccanismo della visione).** *Arch. di Ottal.*, pp. 227-257, September-October, 1938.

(11) **Simonelli** gives an account of the chemistry of the visual purple. He shows that during the bleaching of this, several substances are produced. These have been spectroscopically examined. The original substance, rhodopsin, has an absorption band centred about the length 500 $\mu\mu$; then a substance appears which has an absorption band in the ultra-violet; treated with trichloride of antimony, it becomes blue and has a band of absorption about 620 $\mu\mu$. This is characteristic of vitamin A. A third substance after treatment by trichloride of antimony gives an absorption band about 655 $\mu\mu$. This is a carotinoid whose chemical structure is not yet worked out; it is given the name of retinene. It has been shown that vitamin A is easily derived from carotene. A fourth substance, yellow in colour, giving two absorption bands about 497 and 466, is identified as carotene.

Wald has found that in the chloroform extract of the dark-adapted retina of the frog there is much retinene and traces of vitamin A; in the light-adapted eye there is no retinene but much more vitamin A. Chloroform has a destructive action on the visual purple changing it into retinene. It seems that in the dark-adapted eye, retinene does not exist as such but is combined to some protein molecule. After partial bleaching of the retinal purple, retinene can be found; complete bleaching discloses disappearance of retinene

and appearance of vitamin A in large quantity. When the bleaching is partial producing visual yellow, breaking up the visual purple into retinene and some proteid, reformation of visual purple is easy; when the bleaching has gone on to the formation of visual white, the reformation is much slower; vitamin A must first be reconverted into retinene. This process cannot take place in the isolated retina; the presence of the pigmented epithelium is necessary for this change.

Vercelli discusses the importance of vitamin A in the avoidance of night-blindness. He recalls how this condition may be easily cured by the addition of vitamin A to the food. Recent researches have been made on the changes in the time of dark-adaptation. It has been shown that a large proportion of school children show an increase, which is removed by the addition of vitamin A to their food.

HAROLD GRIMSDALE.

BOOK NOTICE

Physiologic and Clinical Ophthalmologic Problems in relation to Individual Variability. By Professor A. BRÜCKNER, Basle. Reprinted from the *Archiv. of Ophthal.*, Oct.-Dec., 1938.

This brochure comprises lectures delivered in the eye department (Tennent Foundation) of the University of Glasgow in April, 1936. They include problems of colour vision, phenomena of local adaptation, light and dark adaptation, clinical methods (focal illumination, tonometry, sensitiveness of the cornea) and diseases of the eye dependent on constitution (tubercle) and climate.

Of the 99 pages, 73 are devoted to physiological optics. The problems of colour vision are treated from the point of view of the Hering theory, and space is taken up by discussing the exploded hypothesis of the specific brightness of colours and the explanation of the two chief types of colour blindness by differences of macular pigmentation. The almost complete absence of reference to English workers—or indeed any but German workers—is explained by the somewhat naive note: "Special attention was paid to German literature; it was taken for granted that English authors were well known to the listeners, and they were therefore mentioned only in passing." Such a remark does not justify the implied attribution of discoveries to the wrong people—*e.g.*, macular pigmentation to Holm and Kravkov, though it was well known to Clerk Maxwell and Abney; electrical processes in the retina to Kohlrausch and Sachs, whereas the work of Dewar and McKendrick (1873), Gotch,

and above all Granit passes unnoticed. "That colour blindness of the peripheral parts of the retina is only relative was demonstrated by Tschermak, a pupil of Hering, about forty years ago." As a matter of fact it was pointed out by Aubert in 1865.

In spite of these criticisms the brochure contains much of interest to the ophthalmologist.

OBITUARY

A. W. SANDFORD

We regret to record the death of Arthur Wellesley Sandford on May 12. He was born in 1858 in Ireland, the son of the Rector of Clonmel. Sandford was educated privately and at Queen's College, Cork. He graduated M.D., M.Ch. in 1882 and at once began his long connection with Cork as an ophthalmic and aural surgeon. He soon acquired a great reputation all over the South of Ireland and for many years had a very large practice both in private and hospital. He held the post of Ophthalmic and Aural Surgeon to the Cork County Hospital and Southern Infirmary, and on retirement was elected Consulting Surgeon. Sandford joined the Ophthalmological Society of the United Kingdom in 1884, he served on the Council from 1896-1899 and was Vice-President, 1902-1905. On the formation of the ***British Journal of Ophthalmology*** he became our representative in South Ireland and held the post until 1922. He had also served as President of the Irish Ophthalmological Society, and as Professor of Ophthalmology and Otology at University College, Cork.

NOTES

Honour MISS IDA MANN has been elected the first honorary member of the Ophthalmological Society of Australia.

* * * *

Appointment ARTHUR LISTER has been appointed Assistant Surgeon to the Royal London Ophthalmic Hospital.

Royal Society of Medicine, Section of Ophthalmology

THE following officers have been nominated for the season 1939-1940. President, Malcolm Hepburn; Immediate Past President, W. H. McMullen; Vice-Presidents, P. G, Doyne, Percival J. Hay, T. Snowball, A. E. Iles, A. F. MacCallan, J. Cole Marshall; Hon. Secretaries, C. D. Shapland, the Hon. G. J. O. Bridgeman. Council: Physicians, S. H. Browning, R. A. Hickling, D. H. Brinton. Metropolitan members: Wing-Commander P. C. Livingston, T. K. S. Lyle, J. H. Doggart, Jean Dollar, E. F. King, Muriel A. Pugh, F. Ridley, R. S. Scott, H. B. Stallard. Provincial members: J. Foster, W. M. Muirhead, T. Colley, R. Beatson Hird, G. T. Willoughby Cashell, E. G. Recordon. Hon. Librarian, Bowman Library, Arnold Sorsby. Representative on Editorial Committee. E. Wolff.

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Industrial Eye Injuries

IT may come as a surprise to the readers of this Journal to know that this subject was to the fore eighty years ago. In a copy of Chambers' Journal, dated June 5, 1858, there is an article entitled "A Plea for the Eyes," which begins with the admirable statement that "The eye of the workman is assuredly one of the choicest of his working tools."

Various recommendations are made for the care of this tool, such as the wearing of goggles or spectacles of wire gauze, but the most interesting comes from a Dr. Gibb who is "quite certain that many, in fact a large number of artisans who are exposed to the influence of dust, chips, splinters, etc., from the nature of their occupation, suffer more in proportion to the absence of beards and whiskers than those who possess those appendages." He attributes this to two causes; the protection afforded to the face by the hair, the strengthening and tonic influence imparted in consequence to the nerves of the face and eyes, and the general improvement of the health from the comfort experienced in wearing the beard. The second cause is the arrest of particles of dust and grit by the hair of the beard and whiskers.

Whether it would be possible in these days of cheap safety razors to persuade a workman to grow a really good beard is doubtful, but it is conceivable that a "king beaver" might find that he was afforded some protection from particles flying up into his eyes when engaged on work below eye level.

Perhaps there are still some hardy Northerners who retain what nature meant them to have by way of face decoration, and if so it might be worth while to determine whether they were less liable to injuries of the eyes by small foreign bodies than those whose faces were shaved.

Keratitis ramificata superficialis

DR. W. A. WILLE, of Java, whose paper on keratitis ramificata superficialis was published in this Journal in December, 1938 (p. 705), has had enquiries about the fluorescin solution he recommends and has asked us to publish the formula. It is:—

fluorescini, milligram 400.
carbon. kal., milligram 200.
s. in sol. sublimati, 1—10000 gram. 20.

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International Organization against Trachoma

A GENERAL Assembly of Delegates and of all Members of the International Organization against Trachoma, of which due notice had been given, was held at the Royal Society of Medicine, 1, Wimpole Street, London, on Friday, April 21, 1939.

There were present:—Drs. MacCallan (President), Wibaut (Secretary-General and Treasurer), Nordenson and Pfluger (International Council of Ophthalmology), Goodman (League of Nations), Bailliart (International Association for the Prevention of Blindness, also representing France), de Grosz (representing Hungary), Gradle (representing the United States of America), Lauber and Melanowsky (representing Poland), Rohrschneider (representing Germany), Maggiore (representing Italy), Khalil, El Kattan and Tahir (representing Egypt.

The Minutes of the last meeting held in Cairo in December, 1937, were read and approved. The accounts were examined and approved.

The new Statutes, a draft of which had been previously sent to each delegate and member, were approved.

It was decided that the President and Secretary-General should remain in office until the next Concilium Ophthalmologicum, when an election will be held to fill those offices and those of Members of the Council.

A discussion of importance by many members of the Assembly took place. It was decided to delegate Dr. MacCallan to write a "brochure" on the subject of trachoma destined for general practitioners in trachomatous countries. Dr. Gradle suggested that the cost of the necessary illustrations might be borne by certain Corporations which he proposed to interest in this project.

The next meeting of the Council of the Organization was decided on; this will be held in Paris at the same periods as those of the International Council of Ophthalmology and of La Société Française d'Ophtalmologie.

The next Scientific Meeting of the Organization will be held during the period of the next Quadrennial Congress of the International Ophthalmological Congress at Vienna in 1941.

We have received from Dr. Redslob, of the Faculty of Medicine of Strasbourg, a letter expressing profound sympathy on the occasion of the loss of the submarine Thetis. He writes: "La tristesse et la pitié se livent sur la physionomie de tous, grands et petits, mêmes des plus humbles. Nous éprouvons pour les victimes, morts d'une façon si tragique, pour leurs familles, pour l'Angleterre toute entiere, qui perd ainsi une centaine de héros, la plus sincère commiseration." We are deeply grateful to Dr. Redslob for his letter, and take the opportunity of conveying to all our French colleagues our distress at the loss of the submarine Phœnix.

FUTURE ARRANGEMENTS

1939

July 6-8.—Oxford Ophthalmological Congress, at Oxford.

October 6.—Midland Ophthalmological Society, at Birmingham and Midland Eye Hospital. (Annual.)

CONTEMPORARY OPHTHALMIC LITERATURE

Archives of Ophthalmology. *May, 1939.*

REBER and SCHEIE. Bilateral endophthalmitis complicating pneumococcic septicaemia. Report of a case.

BRALEY. The Rickettsia question in trachoma. 1. Microscopic observations on the virus.

PINCUS Naevus flammeus associated with glaucoma: report of a case.

HAGEDOORN. Angioid streaks.

ELWYN. Circulatory disturbances in retina in arteriosclerosis and in essential arterial hypertension.

RAND, IRVINE and REEVES. Primary glioma of the optic nerve.

MCDONALD and PETTIT. The production of corneal ulcers in the rabbit.

HELFAND. Congenital familial external ophthalmoplegia without ptosis, with a lesion of the pyramidal tract.

THEODORE and LEWSON. Bilateral iritis complicating serum sickness.
RUNDLES. Streak retinoscopy.
KRAVITZ. Hodgkin's disease of the lid: report of a case.

Annales d'Oculistique. *April, 1939.*

VEIL and SARRAZIN. Hereditary and familial megalocornea.
RISER, COUADAU, PLANQUES and VALDIGUIE. Ophthalmoscopy in hypertensive disorders.
URECHIA, VANCÉA and DRAGOMIR. The Laurence-Moon-Bardet-Biedl syndrome.
BIRO. Endocrinological connections with retinitis pigmentosa.
CORNET. Interstitial keratitis.

May, 1939.

MORAX. Paralysis of the associated movements of the eyes.
SCHOUSBOÉ. Some manifestations observed in North Africa of hereditary and acquired syphilis at the corneo-scleral level.

Archiv. für Ophthalmologie. *April, 1939.*

KYRIELEIS. Clinical and anatomical observations on albuminuric retinitis.
MÜLLER. The absorption of glucose and ascorbutic acid in the lens.
KREIKER. Stabilisation, a complement to higher science.
ROSE. Blue sclerotics, fragilitas ossium and defective hearing.
SIEGERT. The clinical and serological diagnosis of gonorrhoeal iritis.
RADNÓT. The pathology of orbital lymphangioma.
RAUH. Tetany and cataract under the influence of AT_{10} Holtz (Dihydrotachysterin).
FEHRMANN. Rubeosis iridis diabetica and its general medical importance: with anatomical findings.
WECKERT. Double-eye.
OHM. Nystagmus.
CONTINO. The eye of the argyropelecus hemigymus.
BAKKER. Reply to Wagner's remarks on my experimental infra-red cataract.

Klinische Monatsblätter für Augenheilkunde. *May, 1939.*

FRANCESCHETTI and STREIFF. The relation between retinal artery pressure and the general blood pressure in diabetes.
VOGT. Practical importance of arteriography in ophthalmology.
GOEDE. X-ray photography of the skull in ocular disorders (especially in the calcification of the internal carotid artery).
FISCHER. The question of permanent papilloedema.
KORTE. Contribution to the knowledge of glaucoma.
URBANEK AND ROSCHKOTT. The influence of the seasons and of the weather on the frequency curve of scrophulous kerato-conjunctivitis.
FRIEDE. Penetrating optical keratoplasty with subsequent lens extraction in maculae corneae juvenilis and excessive myopia.
SZINEGH. The use of p-Aminobenzolsulfamid in ophthalmology.

Ophthalmologica Zeitschrift für Augenheilkunde, *May, 1939.*

KREIKER. A simple lid plastic operation for cicatricial entropion.
WEVE. Congenital aphakia with hyaloid artery and retinal fold.
WEEKERS. Advancement or recession in the operative treatment of strabismus? A procedure of recession.
VON BISTIS. Statistical observations on blindness in Greece.

Annali di Ottalmologia e Clinica Oculistica. *February, 1939.*

NICOLATO. Considerations of some results of reconstruction of the lids following the excision of tumours or of severe traumata.
BUCALOSSI. Appreciation of the polychromatic stimuli and the physical and physiological causes of the imperfect discriminative powers of the eye.
TALIERCIO. Irradiation cataract.
RUBINO. Beneficial effects of intravenous iodic therapy in some affections of the visual pathway.
LAGOMARSINO. Bee venom in the cure of trachoma.

Bollettino d'Oculistica. *February, 1939.*

FALLICA. Changes in the curvature of the anterior surface of the cornea by contraction of the extrinsic muscles.
CIOTOLA. Experimental research about the influence of ascorbutic acid (vitamin C) in morbid ocular processes of an anaphylactic nature.
DI BARI. Tolerance in the determination of the axis of the cylindrical lenses employed in the correction of astigmatism.
RASI and GRANDI. Orbital chloroma.
SIMONELLI. The connection of the glucose compensation with the pathogenesis and post-operative course of cataract.

March, 1939.

GRANCINI. Oculo-glandular syndrome (Parinaud's conjunctivitis) of the nature of Pfeiffer's "Bacterium pseudotubercolosis rodentium."
PERSICHETTI. The haemoclassic reaction in lues with ocular manifestations.
ALIQUÒ-MAZZEI. Disciform keratitis from a virus vaccine.
DI BARI. Tolerance of the axis of spectacle lenses for near work.
RUBINO. Large intra-retinal cysts.
GRANDI. A case of disciform keratitis favourably treated with vitamin B1.
MISTRETTA. Epithelioma of the limbus.

Rassegna Italiana d'Ottalmologia. *January-February, 1939.*

TRISTAINO. A case of "elongatio canaliculi lacrymalis inferior congenita."
CECCHETTO. Paracentesis of the anterior chamber in various ocular disorders.
PERGOLA. A case of "Bottone d'Oriente" of the pre-lacrymal region.
MORETTI. Contribution to the surgical cure of trachoma.
VALERIO. Contribution to the study of the surgical treatment of ptosis with particular regard to the introduction of Nida's technique.
BORSELLO. Circumscribed hyperplasia of the palpebral conjunctiva, a papillomatous type, with histological picture similar to trachoma.